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TUBERCULOSIS IN AUSTRALIA.[1]

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IN this address I propose to review some of the more interesting and important aspects of tuberculosis as it has occurred in Australia, and to indicate my views as to the principal measures which should be adopted in order to control this disease in Australia and finally to eradicate it.

Before proceeding with my paper I should like to take the opportunity of thanking the President of the Congress for the honour done to me in inviting me to speak on this subject, an honour which I very greatly appreciate.

[1] This paper is based on one read at a plenary meeting of the Fifth Session of the Australasian Medical Congress at Adelaide, August, 1937.

Tuberculosis in Australia has a most interesting and informative history. In the time I have, I can only briefly touch on a few of the more important aspects.

Preliminary Discussion.

Prior to about 1865 the statistical figures give no reliable index of mortality from tuberculosis because this disease was not differentiated from a number of others in the mortality tables. In Victoria, for example, prior to 1862, phthisis was placed in a group with pneumonia, bronchitis and other diseases of the respiratory system. This is more or less true of other States also. Consideration of statistics from 1865 onwards shows two distinct periods: (i) a period of about 20 years, 1865 to about 1887, in which the death rate from pulmonary tuberculosis was steadily and rapidly rising; (ii) a period from about 1887 to the present day in which the death rate from pulmonary tuberculosis has steadily fallen except for one or two temporary upward tendencies, notably about 1908 and 1918, for which

adequate explanations are available, the temporary upward tendency about 1918, for example, being probably associated with war disabilities.

TABLE I.

Mortality Rates per 100,000 of Mean Population from Tuberculosis of the Respiratory System in the States of Australia.

(Average annual rates for five-year periods.)

Five-year Period.	Rate.					
	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.
1870–1874	*86·4*	121·7	104·9	85·1	*90·0*	97·2
1875–1879	100·7	131·1	135·8	101·6	85·4	100·8
1880–1884	*112·4*	139·8	*166·2*	105·6	88·2	*105·8*
1885–1889	103·8	*145·2*	147·0	*107·0*	88·7	99·7
1890–1894	89·2	135·3	116·7	98·6	83·1	91·4
1895–1899	80·0	122·2	92·2	89·4	65·5	73·0
1900–1904	84·3	114·5	83·7	82·7	76·0	60·8
1905–1909	67·5	95·4	65·6	86·6	78·8	68·8
1910–1914	63·9	77·7	52·1	72·0	70·6	60·8
1915–1919	58·1	67·4	50·8	75·4	77·1	52·6
1920–1924	51·2	62·0	42·1	65·8	70·5	56·4
1925–1929	46·7	54·6	39·0	55·4	65·4	50·8
1930–1934	38·5	44·9	34·6	44·5	50·0	46·6
1935–1936	35·6	41·3	31·6	36·9	45·1	47·0
The Rates of 1935–1936 shown as Percentages of the Rates in 1880–1884.						
1880–1884	100	100	100	100	100	100
1935–1936	32	30	27	35	51	44

The figures in Table I indicate this rise and fall in the mortality rates from pulmonary tuberculosis. The mortality rates given are the mean rates for five-year periods from 1870 to 1934. It will be noted that the apex of the mortality curve is not reached at exactly the same time in each State. For example, the apex in New South Wales was reached in 1880-1884, and in Victoria in 1885-1889, while in Western Australia no early rise in the mortality rate is recorded.

The mortality curve for tuberculosis other than that of the respiratory system (Table II) shows a similar rise followed by a steady fall.

TABLE II.

Mortality Rates per 100,000 of Mean Population from Tuberculosis other than Tuberculosis of the Respiratory System in the States of Australia.

(Average annual rates for five-year periods.)

Five-year Period.	Rate.					
	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.
1870–1874		34·3		17·5		
1875–1879	41·3	*38·4*	25·1	20·4		
1880–1884	32·1	33·2	34·6	*22·7*		7·8
1885–1889	*42·8*	37·3	*63·4*	16·4		10·7
1890–1894	31·7	34·1	46·0	19·8	8·2	14·5
1895–1899	25·6	31·2	34·3	17·8	14·3	*22·4*
1900–1904	17·5	29·7	29·2	14·3	11·7	21·0
1905–1909	15·4	23·1	10·8	13·7	11·8	16·4
1910–1914	10·2	14·5	7·2	14·7	9·6	15·2
1915–1919	8·0	13·0	4·7	13·5	8·1	15·8
1920–1924	6·7	12·0	3·4	9·7	6·7	14·4
1925–1929	5·9	9·1	2·7	8·7	6·8	12·5
1930–1934	4·2	7·7	2·6	6·2	4·9	9·2
1935–1936	3·5	6·1	2·3	5·3	3·5	10·4

Reference to Table I will show that the figures in the several States vary in the extent of total decline in the mortality rate. The greatest decline has been in Queensland, the present rate being 27% of the 1880-1884 rate. At the present time Queensland has the lowest mortality rate from pulmonary tuberculosis in Australia. The very high mortality rate from about 1880 to 1890 in Queensland was largely influenced by a heavy mortality from acute tuberculous infection among Kanakas.

In Western Australia there is the least decline in the mortality rate, the present rate being 51% of the 1880-1884 rate.

Tasmania has the highest mortality rate from tuberculosis in Australia at the present time.

Turning to Table II; showing the mortality rates of tuberculosis other than that of the respiratory system, we find a similar early rise in the mortality rates followed by a steady and pronounced fall. The maximum was reached in the different States at different times. This table is considerably affected by variations which took place in the nosological classification. The several States have not at all times adopted the same classification, consequently, deaths which were attributed to tuberculosis in some States were not in the others. It was not till 1882 that the discovery of Koch's bacillus enabled lesions due actually to tuberculosis to be differentiated from non-tuberculous lesions.

Not only did different States adopt different nosological classifications, but in any given State variations in the nosological classification have occurred from time to time. As late as 1922 the system of classification was altered so that deaths from acute miliary tuberculosis, which had previously been included among deaths from tuberculosis of the respiratory system would be thereafter designated acute disseminated tuberculosis, and would be included in deaths from tuberculosis other than of the respiratory system. This means that from 1922 onwards the deaths attributed to tuberculosis other than of the respiratory system, were considerably augmented by the addition of the deaths from acute miliary tuberculosis. This has had the effect of keeping up the mortality rate from tuberculosis other than that of the respiratory system to a level some 15% higher since 1922 than would have been the case if these deaths had remained attributable to tuberculosis of the respiratory system.

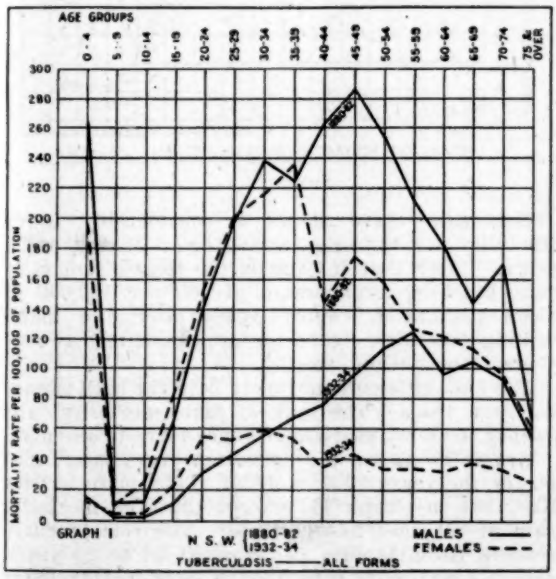
I mention these matters to indicate that the compilation of Table II has presented difficulties and that the figures, particularly for those years prior to 1910, must not be taken as strictly accurate. The mortality rate from tuberculosis other than that of the respiratory system in the earlier decades was probably higher than the figures in Table II indicate, and the fall in the mortality rate has consequently been even greater than would appear from the figures shown in this table. In the several States there are marked differences in the decline of the mortality rate from tuberculosis other than that of the respiratory system. It will be noted that in Queensland the fall is from 63·4 per 100,000 in 1885-1889 to 2·3 per 100,000 in 1935-1936, the latter rate

being only about 3% of the 1885-1889 rate. The Queensland rate is now probably the lowest in the world. In Tasmania, on the other hand, the rate at the present day is still high, and is nearly 50% of the maximum rate. This comparatively high mortality rate from non-respiratory tuberculosis is one of Tasmania's problems, and I shall return to this point later when discussing the type of infection in Australia.

As already stated, Tables I and II show an initial rise in the tuberculosis mortality rate, both from pulmonary tuberculosis and from other forms of tuberculosis, from about 1870 onwards, followed eventually by a steady fall.

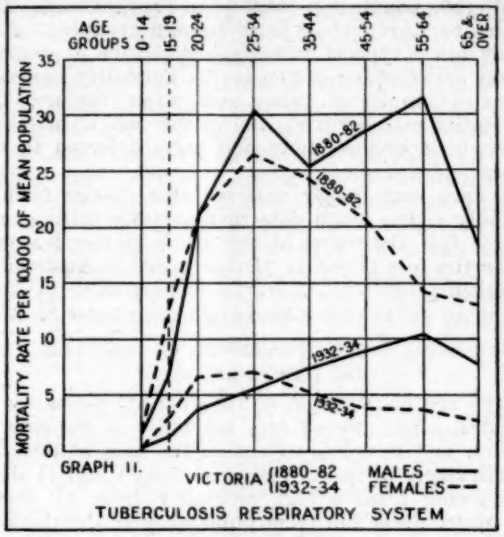
The Rise in the Tuberculosis Mortality Rate.

The rise in the death rate during the period of ascent was largely due to an increased mortality among young adults, which was most pronounced in the case of pulmonary tuberculosis in the years 1870-1885. Graphs I and II indicate the extent of this mortality among young adults in New South Wales (all forms of tuberculosis) and Victoria (pulmonary tuberculosis). It will be noted (Graph I) that the mortality among young adult males, which in New South Wales in 1880-1882 reached 240 per 100,000 of population in the age-group 30-34 years, has now been largely replaced by a mortality which reaches its maximum among males of about 50 to 60 years of age. The high mortality in the age group 0-4 years, both in males and females, will also be noted.



Graph I.

In Victoria (Graph II) the mortality from pulmonary tuberculosis in males, in 1880-1882, almost reached its maximum in the early age group of 25-34 years (305 per 100,000 of mean population). The mortality rate in this age group today is 55 per 100,000. As in New South Wales, the main mortality in males is now in the late age groups. Both graphs show that the mortality rate of females in the early age groups is at present considerably higher than that of males.



Graph II.

In the latter decades of last century there existed in Australia, in addition to the imported more chronic type of tuberculosis, a local variety of tuberculosis, acute in type, and attacking and killing young adults. There is definite contemporary written evidence of this. A factor which was probably of the greatest importance in connexion with the rising mortality rate was the increased birth rate following the introduction into Australia of large numbers of young adults, male and female immigrants, which occurred between 1850 and 1865. The decade 1850 to 1860 saw the greatest proportional increase in population that has ever occurred in Australia. The huge influx of young adult immigrants led to an abnormally high birth rate during that period, and consequently an abnormally large number of persons fell some 20 years later (1870-1890) into the age-group in which mortality from the acute type of adolescent tuberculosis was high. This high death rate from an acute type of tuberculosis was associated with a considerable degree of privation and exposure and physical stress, owing to the conditions of life at the time in a new country. So the mortality rate from tuberculosis rose as has been shown.

The Decline of the Tuberculosis Death Rate.

From about 1890 onwards the death rate gradually fell.

In 1882 the discovery by Koch that tuberculosis was due to the implantation of a specific pathogenic bacillus in the body led to the establishment of laboratory facilities for diagnosis and to the awakening of public apprehension, resulting in the introduction of precautionary measures.

The decade from 1880 to 1890 was the period in which active sanitary reform began in all States in Australia in response to the activity in England.

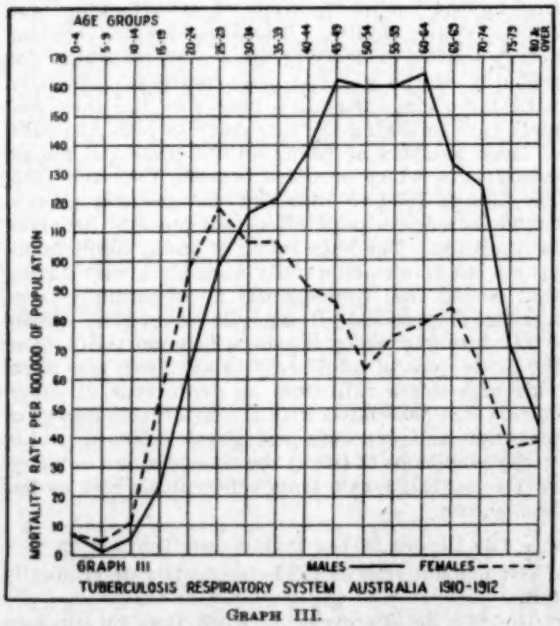
The proportion of young adults in the Australian population became less marked as time went on and the population began to approximate the standard European type in its age grouping. The early acute type of tuberculosis became a progressively diminishing factor in the mortality figures.

As a result of these and other factors, the mortality rate declined, and in the case of males in particular, became more and more deferred to the later age groups.

I have very briefly outlined this change from a rapidly rising death rate to a steadily falling one. For a full discussion of the whole matter I would refer you to a paper on "Tuberculosis in Australia", published by Dr. J. H. L. Cumpston[1] in THE MEDICAL JOURNAL OF AUSTRALIA of August 8, 1931.

THE AGE AND SEX DISTRIBUTION OF TUBERCULOSIS MORTALITY IN AUSTRALIA.

The age distribution of tuberculosis mortality in Australia has altered very markedly in the case of males, and to a less extent in the case of females, particularly in recent years. Graphs I and II show the swing from a high mortality from all forms of tuberculosis and from pulmonary tuberculosis in adolescence and early adult life, to a mortality which reaches its maximum in the case of males at about 50 to 60 years, but in the case of females at about 25 to 35 years.

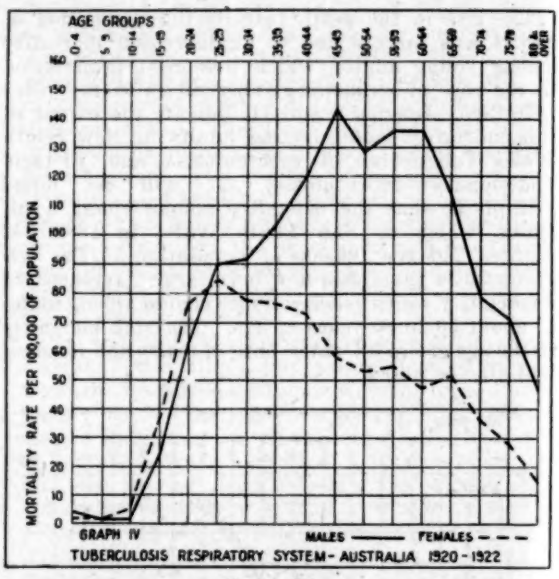


GRAPH III.

The behaviour of the mortality curve varies somewhat in detail in the different States; but the general tendency is the same in each State. Graphs III, IV and V indicate the mortality rate for tuberculosis of the respiratory system throughout Australia. The graphs have been prepared from the mean of the mortality rates over three years, the census year and the one before and after it. The effect of any unusual fluctuation in any one year is thus smoothed out.

The Commonwealth Statistician began to publish his demography tables in 1908, and the figures from year to year are strictly comparable from that date.

The mean curve for the three years 1910 to 1912 (Graph III) shows the mortality rate in females falling from 6 per 100,000 in the age group 0-4 years, to 4 per 100,000 in the age group 5-9 years, then rising to reach a maximum of 118 per 100,000 of mean female population in the age group 25-29 years, and then immediately falling away.



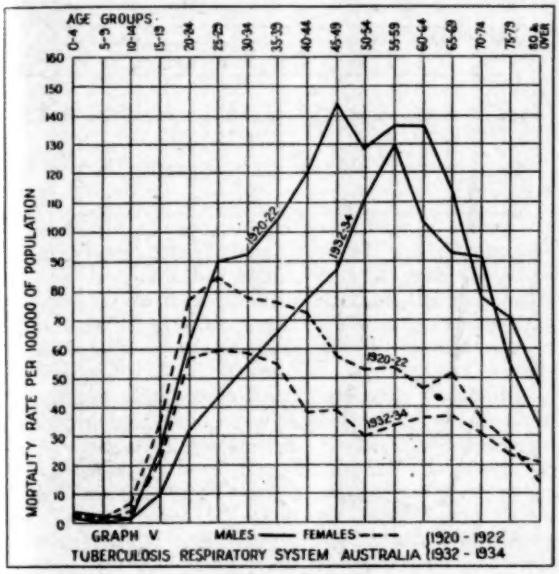
GRAPH IV.

The male curve shows a considerably lower mortality rate than the female for all ages up to 29 years. After that it exceeds the female mortality rate, reaching a maximum of 165 per 100,000 of mean population twenty years later and maintaining this high figure for approximately twenty years, then falling steeply.

The bulk of female mortality is in the ages twenty to forty years. The bulk of male mortality falls twenty to thirty years later (ages forty to seventy).

Graph IV shows the curve ten years later, that is, for the years 1920 to 1922. This curve is very like that in Graph III except that the mortality rate at all ages has declined. The female curve reaches its maximum as before at 25 to 29 years, and the maximum rate, though considerably lower than that of ten years previously, persists from age 20 to age 45 years. The male curve reaches its maximum at 45 to 49 years and is maintained near this level for about 25 years. The female maximum has, however, fallen from 119 to 85, and the male maximum from 165 to 144 per 100,000 of mean population.

Coming to the last census, 1933, we find (Graph V) that the mean mortality rate for the three years 1932-1934 for both sexes has again fallen in every age group. The female mortality curve still reaches its maximum at age 25 to 29; but it shows a tendency to concentrate even more into the earlier age groups than was the case in the previous decade. The apex is almost reached at age 20 to 24 years, and as early as age 40 years the mortality rate drops steeply. The male curve, on the contrary, shows a pronounced slide to the right, indicating a greatly reduced mortality rate in all age groups up to 55 years. The apex is reached ten years later than in the 1920 to 1922 curve, and



GRAPH V.

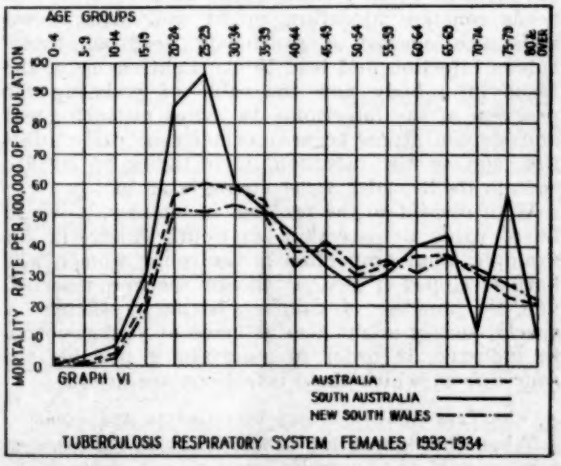
the maximum mortality is maintained for only five years instead of twenty years. In all age groups up to 50 years, the decline in the male mortality rate has been very much more pronounced than in the female mortality rate. To illustrate this, the male and female curves for 1920-1922 are superimposed in Graph V for ready comparison with the curves for 1932-1934. It will be noted that, for the age period 20 to 24 years, the male mortality rate has fallen in ten years from 62 to 31 (50%). In the age period 25 to 29 years, it has fallen from 90 to 43 (52%), and for the age period 45 to 49 years, from 144 to 86 (41%).

It will be noted also that whereas in 1920-1922 the male mortality rate in age group 25-29 years (90 per 100,000) was higher than the female (85 per 100,000), it is now (1932-1934) very much lower than the female rate. The female mortality rate in this age group has fallen from 85 to 60 per 100,000, a decline of 29% in ten years. The male mortality rate has fallen in the same period from 90 to 43, a decline of 52% in ten years.

The female mortality curve remains highest in early adult life and shows none of the tendency of the male curve to slide into the late age groups; that is, the mortality of females from tuberculosis remains largely a mortality of young females. I shall return to this in a moment.

A number of reasons might perhaps be brought forward to explain the very pronounced decline in the tuberculosis mortality rate in males in all age groups up to 55 years. I think it might be reasonably deduced that the action which has been in progress now for many years to control the transmission of infection and to raise resistance against the disease has had the effect of reducing the number and aborting the progress of infections in the early years of life, and so of depleting the reservoir from which mortality would occur later. That is, two principal factors are at work: (i) a diminishing infection rate, (ii) an improving resistance rate. These factors in my opinion account largely for the rapid decline in the mortality rates seen at all ages up to 55 years. The male mortality about the peak period, age 55 to 59 years, might be looked upon as due to a considerable extent to the dying off of the old infections. If so, the curve should flatten considerably in the next ten years, with an apex at an even later age group than 55-59 years.

The fact, however, remains that, in contradistinction to the male mortality curve there is still a preponderance of the female deaths in the earlier age groups. This is brought out strikingly in Graph VI, which shows the female mortality rate in age groups for Australia, New South Wales and South Australia for the three years 1932-1934. The rates are the mean for the three years (the census years and the year before and the year after it), so that any unusual fluctuation in any one year is



GRAPH VI.

smoothed out. This graph shows: (i) The apex of the curve for females as occurring in age group 25 to 29 years in Australia and South Australia, but in age group 30 to 34 years in New South Wales. (ii) A rounded curve for New South Wales and Australia with the peak mortality maintained from about age 22 to 40 years. (iii) A very much more

pronounced death rate from tuberculosis in young females in South Australia rising to 96 per 100,000 in age group 25 to 29 years, as compared with 60 per 100,000 for Australia and 50 per 100,000 for New South Wales. (iv) A steep decline in the mortality rate in South Australia after age 30 years.

The mortality rate among females in age group 25 to 29 years in South Australia is almost double that in New South Wales for the same age group. The mortality rate in age group 20 to 24 years in South Australia (85 per 100,000) is about 40% greater than in New South Wales (51 per 100,000 for the same age period).

After age thirty-four years the female mortality curve in South Australia fairly closely follows that for Australia and New South Wales. It would appear that the extremely high mortality rate in young females in South Australia is a special characteristic of the South Australian mortality curve and is one which urgently requires investigation.

It is thought that the high mortality in young females which occurs notably in South Australia, and also, though to a less extent, in all the States of Australia, is probably associated with two main factors.

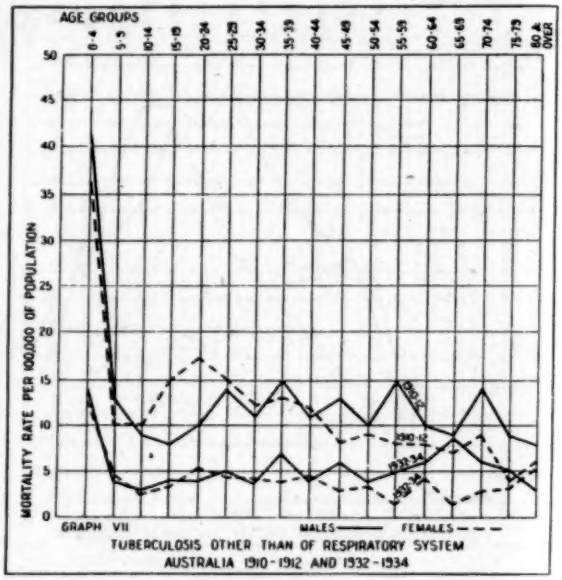
It will be conceded, I think, that tuberculous infection, when it does occur, probably takes place early in life and usually in childhood. It tends to remain latent until some severe stress is placed on the human body, some illness causing the general breakdown of the resistance, or some other special strain, as for example that associated with certain industrial occupations. In females the bearing of the first child, the added domestic cares followed by the birth of another child while the first child still needs constant attention, might well break down resistance against a previously acquired tuberculous infection and lead to the lighting up of the focus in a body now incapable of resisting the progress of the infection. In males no such great and sudden stress occurs normally in early adult life, and so the infection, if it lights up at all, reveals itself under some stress later in life.

With respect to the problem in females, it would be of value to ascertain, in South Australia for example, what proportion of the young women who have developed or died of tuberculosis were married, and the number of children borne by them. A special survey might also be made of young women in industry, in order to ascertain if possible the source from which these infections are arising.

The Type of Tuberculous Infection in Australia.

When the figures for tuberculosis of the respiratory system in Australia are set out against the figures for tuberculosis other than that of the respiratory system, it becomes obvious that in Australia the mortality in children under the age of ten years is accounted for almost entirely by non-respiratory tuberculosis.

Graph V shows a very low rate of mortality in tuberculosis of the respiratory system in ages 0-10 years, beginning to rise at age 14 and rising earlier in females than in males. Graph VII shows the position in relation to tuberculosis other than that of the respiratory system. Here in 1910-1912 the male rate at age 0-4 years was 42 and the female 37 per 100,000. In 1932-1934 the mortality rate in this age group had fallen to 14 (male) and 13 (female). The rate falls rapidly to age 10 years both in males and females, and thereafter through all age groups remains low. It will be noted that there has been a great decline in the mortality rate in all age groups since 1910-1912. The mortality rate which in young females (10 to 30 years of age) was in 1910-1912 considerably higher than in young males is now (1932-1934) below the male rate.



GRAPH VII.

The mortality in children under ten years of age from tuberculosis other than that of the respiratory system has been looked upon by some as being due principally, if not entirely, to infection with the bovine strain of tubercle bacillus contracted through the milk supply. Although this may be to some extent true in certain other countries, it is not so in Australia, where the mortality is almost exclusively due to tuberculosis of human origin.

A comparison will be useful between tuberculosis mortality in Australia and that in certain other countries in which bovine infections are a more important factor than in Australia. In Table III the mortality rates are shown for tuberculosis of the respiratory system in Australia, England and Wales, Scotland and New Zealand from 1881 to 1935. The rates shown are the mean rates for five-year periods. It should be noted that the decline in the mortality rate in Australia is only slightly more marked than in England and Wales, and is actually less marked than the decline in Scotland for the same period.

TABLE III.

Mortality Rates per 100,000 of Mean Population from Tuberculosis of the Respiratory System.

(Average annual rates for five-year periods.)

Five-year Period.	Rate.			
	Australia.	England and Wales.	Scotland.	New Zealand.
1881–1885 ..	130	183	213	91
1886–1890 ..	121	164	190	84
1891–1895 ..	107	146	175	82
1896–1900 ..	94	132	166	79
1901–1905 ..	89	122	148	70
1906–1910 ..	76	111	131	64
1911–1915 ..	66	105	110	56
1916–1920 ..	62	111	99	56
1921–1925 ..	54	86	81	47
1926–1930 ..	49	77	68	40
1931–1935 ..	40	67	59	33
1936	37			
The Rates of 1931–1935 shown as Percentages of the Rates in 1881–1885.				
1881–1885 ..	100	100	100	100
1931–1935 ..	31	37	28	36

In Table IV the mortality rates are shown for tuberculosis other than that of the respiratory system in the same countries and over the same period. Here it will be noted that the decline in Australia is greater than in the other countries, and that in Scotland there is the lowest rate of decline. Scotland, as will be shown, has a comparatively high mortality from human infections of bovine origin, whereas Australia has a low mortality from such infections. Nevertheless in all the countries mentioned the decline in the mortality rate from tuberculosis other than that of the respiratory system is much greater than the decline in the mortality rate from tuberculosis of the respiratory system.

TABLE IV.

Mortality Rate per 100,000 of Mean Population from Tuberculosis other than Tuberculosis of the Respiratory System.

(Average annual rates for five-year periods.)

Five-year Period.	Rate.			
	Australia.	England and Wales.	Scotland.	New Zealand.
1881–1885 ..	34·0	71·0	83·0	
1886–1890 ..	40·0	69·0	71·0	
1891–1895 ..	33·0	66·0	68·0	27·0
1896–1900 ..	28·0	58·0	70·0	24·0
1901–1905 ..	24·0	53·0	70·0	23·0
1906–1910 ..	15·0	46·0	73·0	19·0
1911–1915 ..	11·0	37·0	59·0	14·0
1916–1920 ..	10·0	33·0	47·0	14·0
1921–1925 ..	8·0	22·6	35·8	12·7
1926–1930 ..	6·8	17·4	28·2	9·2
1931–1935 ..	5·0	13·8	20·6	8·4
1936	4·4			
The Rates of 1931–1935 shown as Percentages of the Rates of 1881–1885.				
1881–1885 ..	100	100	100	
1931–1935 ..	15	20	25	

Table V shows the position for all forms of tuberculosis. These figures show that with all our advantages of climate, environment and nutrition in Australia we cannot point to any greater rate of reduction of tuberculosis mortality in this country than has taken place during the same time in England or Scotland. We can, however, point to a lower actual mortality rate from tuberculosis.

TABLE V.

Crude Mortality Rates per 100,000 of Mean Population from All Forms of Tuberculosis.

(Average annual rates for five-year periods.)

Five-year Period.	Rate.			
	Australia.	England and Wales.	Scotland.	New Zealand.
1881–1885 ..	164·0	254·0	296·0	
1886–1890 ..	161·0	232·0	261·0	
1891–1895 ..	140·0	212·0	243·0	109·0
1896–1900 ..	122·0	191·0	236·0	102·0
1901–1905 ..	113·0	174·0	218·0	94·0
1906–1910 ..	92·0	157·0	204·0	84·0
1911–1915 ..	77·0	141·0	169·0	70·0
1916–1920 ..	71·0	145·0	146·0	70·0
1921–1925 ..	62·0	108·0	116·0	60·0
1926–1930 ..	55·4	94·0	96·0	49·0
1931–1935 ..	45·0	81·0	80·0	41·0
1936	41·9			
The Rates of 1931–1935 shown as Percentages of the Rates in 1881–1885.				
1881–1885 ..	100	100	100	
1931–1935 ..	27	32	27	

Table VI shows that Australia has the lowest mortality rate in the world from tuberculosis other than that of the respiratory system, and the lowest rate but one from tuberculosis of the respiratory system. New Zealand apparently has a considerably larger amount of non-respiratory tuberculosis than Australia, though it has a lower mortality rate from respiratory tuberculosis.

TABLE VI.

Deaths from Tuberculosis per 100,000 Living in Various Countries.

Country.	Year.	Tuberculosis of the Respiratory System.	Tuberculosis Other than of the Respiratory System.
New Zealand	1933	32	10
Australia	1934	39	5
United States (Registration Area)	1934	51	6
Canada	1933	53	12
Scotland	1934	57	21
Germany	1932	62	13
England and Wales ..	1933	69	14
Irish Free State	1934	89	24
Norway	1932	111	25
France	1931	131	21
Japan	1933	139	49
Finland	1932	191	32

Experience in some other countries, notably in England and Wales and in Scotland, shows that a considerable proportion of tuberculosis other than that of the respiratory system is bovine in origin, the infection being contracted through the drinking of infected cow's milk, containing the bovine strain of bacillus.

It is important to determine to what extent infection in Australia is bovine in origin. Some work has been done in Australia in typing the strain of bacillus associated with tuberculous lesions in

various parts of the body. Practically all the work in this connexion in typing the strain of bacillus in lesions of children has been done in Melbourne, by Dr. W. J. Penfold, prior to 1923 and subsequently by Dr. Reginald Webster. Dr. Penfold[2] carried out his work on material obtained from the Children's Hospital in Melbourne, and the results of his work were presented to the First Session of the Australasian Medical Congress in Melbourne in 1923. Dr. Reginald Webster,[3] pathologist of the Children's Hospital in Melbourne, also obtained his material from the Children's Hospital and published his results in THE MEDICAL JOURNAL OF AUSTRALIA on March 5, 1932.

The results of Penfold's and Webster's observations have been combined in Table VII. It should be realized that this table refers only to lesions in children, and that the material was obtained from one hospital only, the Children's Hospital, Melbourne. The figures in this table cannot therefore be looked upon as reflecting the true position in all parts of Australia. It will be noted that in bone and joint tuberculosis the human strain of bacillus was isolated in every instance. Due allowance must of course be made for the relatively small number of examinations made. In the investigation of tuberculosis of bronchial glands and lungs and in tuberculosis of the meninges the bovine strain was found in a small percentage of cases. In tuberculosis of the tonsils, cervical glands and mesenteric glands the bovine strain of bacillus was found in the great majority of the cases (approximately 60% to 70%).

TABLE VII.

Strain of Tubercle Bacillus Found in Lesions in Children aged 0 to 16 years, in Melbourne.

Tissues.	Number of Cases Investigated.	Strain of Bacillus Isolated.		Percentage Bovine.
		Human.	Bovine.	
Bone and joint	26	26	0	0
Spleen	9	9	0	0
Bronchial glands and lungs	52	48	4	7·7
Meninges and nervous system	7	6	1	14·3
Tonsils	7	3	4	57·1
Cervical glands	27	9	18	66·6
Mesenteric glands	14	4	10	71·4

Table VIII indicates the results obtained in the typing of the bacillus in material from tuberculous lesions in adults. In this table are combined the results of the work of Dr. W. J. Penfold, who examined seventy specimens of sputum from Bendigo, Victoria; of Dr. Reginald Webster, who examined material from five cases of bone and joint tuberculosis in Melbourne; and of officers of the Commonwealth Health Laboratories, who examined material, mainly sputum, from various parts of Australia. It will be noted that in every instance in adults the human strain of bacillus was isolated.

TABLE VIII.

Strain of Tubercle Bacillus Found in Lesions in Adults in Australia.

	Number of Cases Investigated.	Strain of Bacillus Isolated.	Percentage Bovine.
Sputum	141	All human.	0
Pleural fluid	1	All human.	0
Urine	3	All human.	0
Bone and joint	5	All human.	0
Unspecified	3	All human.	0
Total	153	All human.	0

These results may be compared with those obtained in other countries (see Table IX). It will be noted that the results of typing in Australia accord fairly well with those obtained in the United States of America. In both these countries, apparently, the human strain is invariably found in bone and joint tuberculosis, whereas, as W. G. Savage[4] has shown, in Scotland 50% and in England 18% of such lesions were caused by the bovine strain. In England and Scotland tuberculosis of the brain and meninges is far more frequently caused by the bovine strain than in Australia or the United States of America. In all these countries examination of abdominal lesions and tuberculous cervical glands reveals the bovine strain in a large percentage of cases. Pulmonary tuberculosis everywhere is almost exclusively human in origin.

Table X gives some indication of the proportion of non-respiratory tuberculosis attributable to a bovine source of infection in the several countries. The figures in this table must, however, be treated with caution, because it is obvious that the percentage in the last column could be varied according to whether, for example, material from bone and joint lesions (which invariably contains the human strain in Australia) or material from cervical glands (which usually contains the bovine strain in Australia) were allowed to pre-

TABLE IX.

Proportion of Cases in which the Bovine Strain of Tubercle Bacillus was Found in Persons of All Ages in Various Countries.

Country.	Brain and Meninges.		Bone and Joint.		Abdominal.		Cervical Glands.		Pulmonary.	
	Number Investigated.	Percentage Bovine.	Number Investigated.	Percentage Bovine.	Number Investigated.	Percentage Bovine.	Number Investigated.	Percentage Bovine.	Number Investigated.	Percentage Bovine.
Australia	7	14	26	0	14	71	27	67	154	1
United States of America	35	6	17	0	10	70	54	37	291	0
England	30	27	511	18			112	45	202	1
Scotland	10	30	98	50	8	63	90	86	116	2

ponderate in the typing of the strain of bacillus. Thus in Australia, if the same number of patients with brain and meningeal tuberculosis had been examined as of patients with tuberculosis of the cervical glands, the figure of 37% bovine would have been reduced.

TABLE X.

Percentage of Non-Respiratory Tuberculous Infection in Children aged 0 to 15 years Attributed to a Bovine Source of Infection.

Country.	Number of Cases Investigated.	Percentage Bovine.
Victoria (Australia)	90	37
United States of America	125	25
England	740	33
Scotland	199	68

Penfold concluded from his observations in Melbourne in 1923 that 26% of tuberculous infections in the first four years of life were of bovine origin.

Now, having determined that the bovine strain of bacillus in Australia is found only in children, not in adults, and that it is found in a large proportion of abdominal lesions and in tuberculosis of the tonsils and cervical glands, but rarely in tuberculosis of the meninges, not at all in tuberculosis of bones and joints, and with the greatest rarity in tuberculosis of the respiratory system, let us examine the mortality rates from tuberculosis in these several situations.

It will be noted (Table XI) that in tuberculosis of the intestines and peritoneum, which in 70% of cases is bovine in origin, the mortality rate for the five-year period 1931-1935 is only 19% of the mortality rate of 25 years ago (1906-1910). In all those situations, on the other hand, where infection is usually human in origin, the mortality rate is from 35% to 76% of the rate of 25 years ago. It would appear, then, that in Australia the fall in the mortality rate from lesions due mainly to the bovine strain of bacillus has declined to a very much greater extent than the mortality from lesions due to the human type of bacillus. This indicates that mortality from tuberculosis of bovine origin in Australia is now very low indeed.

For a detailed survey of bovine tuberculosis in man and animals in Australia reference may be made to a report on this subject published by the Commonwealth Department of Health in 1930.[5]

Taking all the factors discussed into consideration we may reasonably conclude as follows:

1. That in Australia the amount of tuberculosis in children attributable to infection with the bovine strain of bacillus is by no means inconsiderable, and that consequently attention must be given to the elimination of sources of bovine infection. The infection is practically confined to children under fifteen years of age.

2. That the mortality from human tuberculosis of bovine origin is now very low in Australia and has declined much more rapidly than the mortality from tuberculosis of human origin. This may be attributed mainly to smaller dosage of infection through the milk supply, since other factors, such as improvement in treatment, environmental conditions and nutrition, apply equally to tuberculosis of human origin. In respect to its low mortality (probably the lowest in the world) from tuberculosis of bovine origin, Australia holds a particularly favourable position as compared with other countries.

3. That the mortality from tuberculosis in Australia is almost exclusively due to the human strain of infection, and that consequently our main objective must be the breaking of the human chain of infection.

4. That the geographical distribution of human tuberculosis of bovine origin in Australia is variable, being probably more in evidence in some States than in others (see Table II).

Attention has already been drawn to Table II and to the fact that in Tasmania the mortality rate for tuberculosis other than respiratory tuberculosis has not fallen to the same extent as in most other States. Here is a problem for investigation. Is this comparatively high mortality rate from non-respiratory tuberculosis in Tasmania due to a greater persistence of bovine infection in that State? It will be necessary to determine where the mortality mainly lies, that is, in what proportion

TABLE XI.

Tuberculosis in Australia, 1906–1936, classified according to organs affected. Mortality rates per 100,000 of mean population.

(Average annual rates for five-year periods.)

Five-year Period.	Tuberculosis of the Respiratory System.	Tuberculosis of the Meninges and Nervous System.	Tuberculosis of the Intestines and Peritoneum.	Tuberculosis of the Spinal Column.	Tuberculosis of the Joints.	Tuberculosis of the Other Organs.
1906–1910 ..	75·7	5·2	3·6	0·89	0·48	1·37
1911–1915 ..	65·9	4·9	2·5	1·06	0·41	0·96
1916–1920 ..	61·6	4·0	2·3	0·98	0·42	0·92
1921–1925 ..	54·2	3·1	1·5	0·86	0·42	0·77
1926–1930 ..	48·6	2·7	1·0	0·67	0·27	0·69
1931–1935 ..	39·9	1·9	0·7	0·68	0·17	0·51
1936	37·5	1·6	0·6	0·70	0·12	0·49
The Rates in 1931–1935 expressed as Percentages of the Rates in 1906–1910.						
1906–1910 ..	100	100	100	100	100	100
1931–1935 ..	53	37	19	76	35	36

it is due to abdominal tuberculosis, bone and joint tuberculosis, tuberculosis of meninges. It will be necessary also to type the bacillus in the various lesions in order to ascertain whether the strain is bovine or human. A contrast will be noted in the very dramatic fall of the mortality from non-respiratory tuberculosis in Queensland. The determination of the reasons for the different behaviour in Tasmania and in Queensland should provide a fruitful field for investigation.

THE ERADICATION OF TUBERCULOSIS.

Now that I have indicated some of the principal points in regard to the behaviour of tuberculosis in Australia it is appropriate to consider what should be done in order to hasten the decline in mortality, and ultimately to eradicate the disease.

The Eradication of Tuberculosis of Bovine Origin.

We have seen that the amount of tuberculous infection in children in Australia from bovine sources is not inconsiderable, although the mortality from such infection is very low in most States. It is necessary, therefore, to eliminate as far as possible infection from bovine sources. We have reason to believe that a comparatively small dose of bovine tubercle bacilli, particularly if frequently repeated, may set up a tuberculous infection in young children. The principal channel of infection is through the milk supply. Meat and cheese present little danger.

From evidence available it can be estimated that about 8% of dairy cattle in Australia are infected with tuberculosis. The proportion, however, varies in different States and different parts of the same State.

Bovine tuberculosis can be eradicated from and kept out of herds, and it is considered that it is a necessary and also an economically sound policy to extend the system of eradication to all dairy herds in Australia, through the wide and controlled use of the tuberculin test and the appropriate disposal of all affected animals. This is the considered opinion of the members of the Australian Veterinary Association, as expressed at their general meeting in Sydney last year.

I would suggest also, not only from the point of view of prevention of tuberculous infection, but the prevention of a large number of other infections seriously affecting children in Australia, that all milk for human consumption should be pasteurized. The National Health and Medical Research Council of Australia, at its last meeting in June of this year, expressed this opinion in the form of a resolution. There is still an impression in many minds that pasteurization affects the nutritive value of the milk. The National Institute for Research in Dairying, England, has this year (1937) issued a comprehensive report embodying the results of extensive research on the effects of commercial "holder" pasteurization on the nutritive value of milk as measured by laboratory methods.[6] This investigation has shown that the nutritional availability of calcium and phosphorus in the milk, and the biological value and true digestibility of the nitrogen are not affected. Vitamin *A* and the provitamin carotene are not affected. There is some loss of vitamins *B* and *C*.

The Eradication of Tuberculosis of Human Origin.

I have endeavoured to show that mortality from tuberculosis in Australia is almost exclusively due to tuberculosis of human origin. Penfold[7] reported to the First Session of the Australasian Medical Congress in 1923 that in 25% of males and 22% of females examined *post mortem* in Australia there were definite tuberculous lesions, and that the *post mortem* frequency of tuberculosis was greatest in the age period 11-20 years. He also reported to the same congress that in several large hospitals in Sydney, Melbourne and Adelaide, the von Pirquet skin reaction indicating invasion by the tubercle bacillus was positive in from 62% to 67% of the hospital population. These were the figures of 15 years ago: 65% of the population invaded by the tubercle bacillus and 24% of the population having definite tuberculous lesions at the time of death.

I think I have given some indication that the rate of actual infection is not so high today. The mortality rate certainly is much lower. Nevertheless, 2,836 persons actually died of tuberculosis in Australia last year, and neither this figure nor the very high death rate which persists among young females can be viewed with any degree of equanimity.

The cardinal facts as stated by Sir Robert Philip, Professor of Tuberculosis, University of Edinburgh, are as follows: (i) That infection is widespread, more or less universal in the community; (ii) that infection is contracted for the most part in childhood; (iii) that, once established, the infection exerts its influence throughout life either by securing protection (immunity) from the consequence of further exposure, or by causing recurrent disturbances: (iv) that in proportion as the resistance is kept nigh, the disturbances are few and slight, and that in proportion as resistance is lowered the infection is apt to be activated and to spread.

These cardinal facts must be the guiding principles of our fight against the disease. The spread of the infection must be controlled. The evidences of infection and the earliest manifestations of the disease must be sought patiently and persistently in all contacts, from childhood upwards. The resistance of the patient and of the contacts must be built up and maintained, so as to obtain immunity if possible. In this way the patient, if the condition is diagnosed early, may be prevented from becoming infective, or infectiveness if present may be rapidly eliminated. The contacts will cease to be exposed to further infection, and, as a result of enhanced resistance, will be able to overcome any infection previously acquired.

For twenty years or more we have known these things. We have had in our hands the knowledge

and means for the gradual eradication of the disease. But the chief weakness in our system is still the weakness of twenty years ago, the disproportionate concentration of attention on the pronounced lesions. Pulmonary tuberculosis is a late visceral manifestation in early adult or later life of an infection contracted much earlier, probably in childhood. Therefore attention must be shifted more to the very earliest manifestations of infection, though not, of course, to the neglect of the later manifestations—the visceral lesions. The first indications of infection may be slight; but they are recognizable. In this stage infection can be checked and eradicated and immunity can be established. The appearance of new foci of infection can thus be prevented. On the other hand, when visceral manifestations have become clinically obvious the chance of cure is relatively small and infection has already been passed on.

In the "Report of the Director-General of Health, New South Wales", 1935,[8] which has just been published, the results are given of an investigation into the after histories of 280 patients admitted to various sanatoria in New South Wales in 1930. These patients on admission were in an early stage, or in some cases in the middle stage of the disease. Of the 280 patients admitted in 1930, 109, approximately 40%, had died within five years. Obviously we must detect the disease earlier than on the appearance of clinical signs of visceral manifestations.

Measures for the Control and Eradication of Tuberculosis.

Administration.

In order that the measures adopted may operate with success it is considered essential that the whole of the activities within each State should be under the control of a State Director of Tuberculosis, who should be engaged full time on the work, should be highly experienced, and should be given every power and facility to coordinate all tuberculosis activities, departmental and municipal, medical and voluntary. He ought, among other powers, to have the direction of the disposal of patients or contacts to sanatoria, preventoria or other institutions, so that these various institutions may be utilized to the best advantage and reserved for the purpose and the special type of case for which they are suited.

The Tuberculosis Clinic or Dispensary.

In order to maintain that persistent search for the earliest manifestations of disease, and so prevent the development of new foci of infection, the clinic or dispensary system is essential. This system must be comprehensive and complete in all its details. Half-measures are of little use. The clinic system must cover the whole range of activities of tuberculosis control. It must act as a centre for the following:

1. The careful investigation of doubtful cases.
2. The classification of cases of tuberculosis, and the making of arrangements, when necessary, for the disposal of patients to suitable institutions.
3. The examination and continued observation of contacts and the detection of tuberculous infection in its earliest stages.
4. The disposal of ill-nourished contacts or those in an unsatisfactory state of health, to preventoria or other institutions, where resistance can be built up.
5. The supervision and after-care of domiciliary patients, including a comprehensive system of home visitation for the regulation of the life and environment of the patient and his contacts, and for maintaining nutrition and building up resistance against infection.
6. The investigation of the financial position of patients by trained nurse inspectors, with the object of making such arrangements as are possible for relief.
7. The continued supervision of patients discharged from sanatoria or other institutions.
8. The rendering at all times to the general practitioners of the district of advice and assistance in connexion with diagnosis or treatment in any case of the disease.
9. The collection of information and records regarding tuberculosis.
10. The education of the patient and of the public.
11. The providing of certain forms of curative treatment in special cases.

The best system would probably be the establishment of one central departmental clinic in each State, conducted by an experienced and full-time officer of the State Department of Health, working directly under the State director of tuberculosis. This clinic could function either in conjunction with a large hospital or as a separate institution with access to a large hospital, the various departments of which (medical, surgical, dental, pathological, laboratory, X ray *et cetera*) would be automatically available for consultation under a smoothly running team-work system.

In addition to the central departmental clinic, there should be a sufficient number of branch clinics situated at strategic points, each operating in association with a hospital. In large cities one such clinic should be available for each 300,000 of population.. In other areas there should be clinics at suitable central points to cover large districts. A mobile chest clinic is desirable to carry the clinic system into country districts and, by arousing interest, pave the way to the establishment of permanent local clinics in those districts.

The chest clinic or tuberculosis clinic system aims at the detection of every fresh case of tuberculosis in its earliest manifestations and the proper care of the patients to prevent their becoming infective to others, or, should they become infective, to prevent the spread of infection from them. Not only is every effort made to get the patient himself under suitable institutional treatment, in order, if possible, to abort the disease, and in order to inculcate the principles underlying the prevention of the spread of infection, but the close contacts are kept under periodical supervision in order to detect the earliest manifestations of incipient disease or the gradual development of a condition of health favourable to the inception of the disease, and to take the necessary precautions to ensure the maintenance of adequate resistance against the disease.

Notification.

To enable this objective to be accomplished the clinics must be brought into the earliest possible contact with existing cases of tuberculosis. Notification of all forms of tuberculosis, whether infective or not, is necessary, and in order that this notification may be made at the earliest possible moment, the clinics must maintain active and close association with the general practitioners in order to obtain their cooperation. It is considered that notification should be made on reasonable suspicion of the disease. Notification must be immediately followed by administrative action and the full application of the clinic system.

Education.

Neither medical practitioner nor patient appears in general to realize the fundamental importance of the earliest possible diagnosis to enable treatment to be undertaken while the patient is curable. With this end in view every endeavour should be made to inform medical undergraduates and practitioners of the objectives of the clinic system and to impress upon them the importance of their active cooperation in the system. There should also be deliberate and continuous education of the public in order that intelligent precautionary measures may be taken and dread of the disease be replaced by a realization of the advantages and importance of the examination, investigation and observation of contacts, and that early detection is an essential element in successful treatment.

Cooperation with Other Activities.

There should be the closest cooperation also between the tuberculosis clinic system and the maternal and child welfare work, the school medical services, local health authorities, voluntary social welfare organizations and other activities in constant touch with the public.

Diagnosis.

Early diagnosis is an essential objective of the clinic system, the end in view being prevention of the transmission of infection either actually or potentially, and the ensuring of a good prognosis. The means adopted are physical examination of contacts, X ray examination of contacts, examination of sputum, and tuberculin testing. These examinations should be periodical and should be especially frequent and careful between the ages of fifteen and twenty-five years. The high mortality in young women of twenty to thirty years sufficiently emphasizes the need to look for early evidence in childhood and early adult life.

The tuberculin skin test is a valuable aid in diagnosis. It gives us information that tuberculous infection has or has not taken place. If the child with a positive skin test remains healthy we may conclude that its resistance is good and that immunization is in progress. If, however, there is evidence of malnutrition or departure from health, this should be the call for active measures, immediate removal from the source of infection, and the development of resistance by all practicable means. The tuberculin reaction can also be used to reduce the number of X ray examinations of contacts, since such examination could be practically limited to those giving a positive skin reaction. It is quite practicable for actual tuberculous infection to be diagnosed before tubercle bacilli appear in the sputum. The skin reaction gives the clue. The X ray examination furnishes the evidence.

Tubercle bacilli appear in the sputum, not continually, but at intervals, as the foci break down. Sputum examination must therefore be persistent, and the fullest facilities for frequent examination should be available to general practitioners. Tubercle bacilli can be discovered with certainty by microscopic examination only when they are present in large numbers in the sputum. The failure to find them in the sputum is therefore not of much diagnostic value. The use of cultural methods overcomes this difficulty, and there is no reason why the cultivation of tubercle bacilli should not be practised just as cultivation of diphtheria bacilli is practised as a diagnostic test. The cultivation of tubercle bacilli now presents little difficulty, and the employment of this method will detect the presence of tubercle bacilli even when in very small numbers in the sputum or other material examined. In the early stages of pulmonary tuberculosis tubercle bacilli are present in small numbers only in the sputum, and this is just the stage when it is most necessary to detect them. Shrewsbury and Barson,[9] of the University of Birmingham, reported in *The British Medical Journal* of June 5, 1937, that of 135 specimens of sputa which were found free of tubercle bacilli under ordinary microscopic examination, bacilli were detected on cultural examination in 29, that is, 21·4%. Shrewsbury and Barson state that the method of culture employed by them yielded 100% of positive cultural isolations from tubercle bacilli in sputum, cerebro-spinal fluid, urine, fæces and pus.

I suggest that the cultivation method should be used as a routine measure in Australia in those cases in which tubercle bacilli are not found on ordinary microscopic examination in the sputum, and that arrangements for this test should be made at the various pathological laboratories and by the various tuberculosis clinics.

Accommodation.

Sanatoria: Australia is fairly well supplied with sanatorium accommodation; but this accommodation is not always used to the best advantage. There is not in all States accommodation in separate institutions for patients with early and patients with advanced disease. Beds are frequently occupied for very long periods by patients whose grade of infectivity is not high and who nevertheless can get very little permanent benefit from institutional treatment. Such patients, after thorough grounding in precautionary measures, could often be treated in their own homes if there were a thoroughly

efficient dispensary system and if there were an adequate pension system. The pension would cost less than the institutional accommodation, which amounts to somewhat more than £3 per week. If sanatoria were relieved of the long residence of these patients there would be sufficient accommodation for suitable patients. It is not thought that much expenditure is needed for the provision of more sanatorium accommodation.

Preventoria: Additional accommodation is needed for the patient who has not developed an active focus of disease, the child, for example, with a positive tuberculin skin reaction, who is ill-nourished or unhealthy, or who for any reason should be removed for a time from any source of possible infection and whose resistance requires building up. Such institutions, known as preventoria, are at present represented in Australia by a very few establishments maintained by voluntary enterprise, as, for example, the Red Cross organization in New South Wales. There should be more preventoria and they should be provided by the State and should be as much a part of the system of control as sanatoria. Tuberculous infection is probably contracted in childhood. It can be readily overcome at this stage, and suitably operated preventoria form essential links in the system of preventing the development of fresh foci of infection in the community.

Village Colonies: Tuberculosis village colonies probably have a less important place in Australia than in more thickly populated countries. They represent a relatively expensive method of handling tuberculous families.

Vocational training, however, has an important place, and coupled with a good pensions system should be capable of overcoming many of the economic and social difficulties of the tuberculosis sufferer.

Nutritional Aspects: Family Infection.

It is in the family environment of the tuberculous patient that transmission of infection is most liable to occur, and for the following reasons.

Under present conditions few persons can afford to be sufferers from tuberculosis in an early stage. For economic reasons such a patient, being able to work, will continue at work as long as possible. When, owing to failing health, he seeks treatment, the prospect of cure is already poor and he has probably already passed the infection on to his closest contacts, those in the family circle. Even when active disease is diagnosed, he will not leave the home to enter a sanatorium. The pension for which he is eligible is 19s. per week. On his entering the sanatorium the State receives 14s. per week and the pensioner 5s. per week. The result is obvious. The patient prefers to remain at home in receipt of 19s. per week rather than go to an institution and lose his pension. The focus of infection is therefore retained in the home. The pension is totally inadequate to provide suitable environment and nutrition for the family. The family is ill-nourished, ill-clothed and ill-housed. The source of infection remains. The resistance of the close contacts exposed constantly to infection is at a minimum. The infection transmitted to them has every prospect of successful implantation and active progress. The condition of the patient himself, ill-nourished and financially worried, becomes worse and his infectiveness increases. Should he go to a sanatorium he remains there for a while, but is then discharged to return home, often still infective or, if not, soon becoming so again under the unfavourable environmental conditions.

The position of the tuberculous ex-soldier and of his family is very much better. The Repatriation Department pays an adequate pension, which continues in full whether the patient is receiving institutional treatment or not. Excellent treatment is provided in the department's institutions. The value of this system is evidenced by the following facts:

1. Because his pension permits of his family's being adequately housed, clothed and nourished, the tuberculous soldier is prepared to enter an institution for treatment and to remain there as long as is necessary.
2. Because his mind is relieved of financial worry he is in the best condition to benefit from this treatment and to have his disease arrested, and thus he ceases to be a source of infection to his family.
3. The experience of the Repatriation Commission in relation to the arrest of the disease in tuberculous soldiers has been more satisfactory than in civilian patients.

Such data as are available show that the rate of infection of contacts of tuberculous soldiers is less than in the tuberculous civilian household. In South Australia, where skin tests were carried out on children at the Adelaide Children's Hospital for two years, it was found as follows:

1. That of children with no history of family contact, 10% reacted and 90% did not react to the skin test.
2. That among children with a history of definite family contact with tuberculosis the percentage of reactors was 60 and non-reactors 40.
3. That of children of tuberculous ex-soldiers 40% reacted and 60% did not react.

The Repatriation Department has now embarked upon a survey of the close contacts of tuberculous ex-soldier patients throughout Australia in order to ascertain the extent to which infection has been passed on to family contacts. The results of this survey will be awaited with the greatest interest and will supply data for comparison with that available from tuberculosis clinics in respect of civilians.

It is obvious, however, that under present conditions the work of tuberculosis clinics is greatly handicapped by the lack of provision for maintaining the nutrition of the contacts of tuberculous patients and for raising and maintaining their resistance to infection, and by the financial worry from which tuberculous patients cannot escape. Under the existing conditions the transmission of infection and the occurrence of new cases to create fresh foci of the infection later on, cannot be

avoided, even under the best conditions of supervision provided by an effective dispensary system. It is considered of the greatest urgency and of the first importance that a system should be established which would provide an adequate allowance for the families of tuberculous patients, and that this allowance should be paid in full for the maintenance of the family, both when the patient is under treatment at an institution and when he is under treatment in his own home.

The question of tuberculosis was considered by the National Health and Medical Research Council at its first meeting at Hobart in February of this year, and this council expressed the opinion that the economic factor was of the utmost importance, and recommended that a pension should be paid to persons suffering from tuberculosis similar to that now paid by the Repatriation Department and Commonwealth Invalid Pensions Department to tuberculous ex-soldiers receiving a service pension. This pension, for a man, wife and four children, amounts to £2 17s. 6d. per week.

SUMMARY.

The principal measures which it is considered should be employed to control and eradicate tuberculosis may be briefly summarized as follows:

1. The appointment in each State of a carefully selected experienced officer as full-time director of tuberculosis, working under the State department of health.

2. The establishment in every State of a central model tuberculosis clinic or dispensary, with branch clinics at strategic points as necessary. The clinics should be based on large hospitals and should cover the whole range of clinic activity. A mobile clinic as a forerunner to the establishment of permanent clinics is recommended for country areas.

3. Compulsory notification of all forms of tuberculosis. Notification to be on reasonable suspicion.

4. Close cooperation between the tuberculosis branch of the State health department and the maternal and child welfare system, education department, hospitals, medical profession and the public.

5. Provision of every facility for early diagnosis, including laboratory diagnosis, available to all general practitioners free of cost. Laboratory diagnosis to include cultural methods.

6. Provision of accommodation for patients with advanced disease to be separate from that for patients with early disease.

7. The establishment of preventoria.

8. The modification and extension of the pensions system in respect of tuberculous invalids, in order to provide an allowance sufficient to enable the family to be properly nourished and the resistance of family contacts to be built up and maintained.

9. Education of medical undergraduates and graduates to appreciate fully the prophylactic aspects and to cooperate actively in the clinic system.

10. Education of the public to allay dread and to stimulate appreciation of the advantages of early examination, observation and supervision, and to encourage the taking of intelligent precautionary measures.

11. Tuberculin testing of dairy herds and elimination of reactors.

12. Pasteurization of milk.

None of these recommendations is new. Many of them appear in the recommendations of the States of Australia Conference on Tuberculosis in 1911 (twenty-six years ago). They are all set out in complete detail in the report on the control of tuberculosis in Australia adopted by the Federal Health Council in 1929[10] and reaffirmed by the same council in 1935. Some action in respect of a majority of these recommendations has already been taken in various States, but incompletely in most instances. To be effective, the system must be applied actively and in its entirety.

CONCLUSION.

Tuberculosis is a disease about which we know a great deal. For many years the causative organism has been known, and it can easily be detected. The method of entry of the disease into the human body and the progress of the disease are understood. Its method of spread is known. Early diagnosis not only is possible, but presents no great difficulty.

The disease, if recognized very early, can readily be cured, and the patient can thus be prevented from becoming a source of infection to others. The resistance of contacts can be built up and maintained so that any invasion, or even incipient infection, can be overcome and the disease prevented from developing, while at the same time resistance against subsequent invasion is increased. With knowledge less extensive than this many diseases in the past have been wiped out.

Sir Robert Philip teaches his students that the best climate for tuberculosis is that which wooes the patient into the open air. This holds for prevention as well as for cure. In this respect Australia is most fortunate. With our great natural advantages in climate and in relatively high standard of living, we can look with nothing but dissatisfaction on the existing mortality rate from tuberculosis in Australia. We perhaps take some pride in the fact that the tuberculosis death rate in Australia is lower than in most other countries. We have no reason for that pride. The rate of decline of the tuberculosis mortality rate in Australia is no more marked than in Scotland, a country with little of our natural advantages in climate and general living conditions. Our preventive measures are incomplete, ill-balanced and piecemeal.

Tuberculosis presents the very best of opportunities for a successful demonstration of the effectiveness of preventive medicine and of the economic value of prevention. We have every requirement for a completely successful outcome to a determined effort to eradicate the disease. We should have no tuberculosis in Australia. This is an ideal and a goal practicable of attainment if we would wholeheartedly adopt the measures ready to our hand. On the other hand, failure to develop the prophylactic aspects and persistence in concentrating our main attention on the attempted cure of the relatively incurable pronounced lesions can result only in persistence of infection and a continuous procession of new foci of infection.

It is admitted that eradication cannot be attained without considerable expenditure. But let us look briefly at the economic aspect. The cost to Common-

wealth and State Governments in pensions for tuberculosis and in treatment and prevention of this disease is more than one million pounds *per annum*. If tuberculosis mortality had continued at the same rate as in 1880, this governmental expenditure would now exceed five million pounds *per annum* instead of one million. Therefore the success already attained in fighting tuberculosis is saving Australia millions of pounds every year.

The eradication of the disease, which is a practicable ideal in this country, would save millions of pounds in the future. I believe that measures directed to the total eradication of the disease would cost less over the next twenty years than is spent at present in partially checking the disease. We cannot hope for eradication in a few years. In patients already infected the disease will be developing for many years to come. But we can very greatly hasten the decline in the mortality rate, and we can, by effectively applying known methods of control, look forward to virtual eradication within a generation.

The success already attained by our incomplete and ill-balanced methods is an earnest of the infinitely greater success which would follow the persistent application of wholehearted measures. We should aim at nothing less than a Commonwealth free of tuberculosis.

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SOME COMPARATIVE ASPECTS OF TUBERCULOSIS IN LOWER ANIMALS.[1]

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THERE is nothing very new to be said on the subject of tuberculosis in lower animals, particularly in its relationship to the disease in the human race; but the subject of tuberculosis in man cannot be adequately dealt with by the physician and the public health officer unless some consideration be given to the knowledge gained by study of the disease in the lower animal.

In this respect it is tuberculosis of the domestic animals that is of most interest and importance. All the domestic animals are susceptible to the disease, which varies in frequency in the different species. Man may become infected by the use of animal products as food, or he may himself infect one or more species, which in turn may spread the disease among members of their own species as well as among members of human society.

Tuberculosis is most common in cattle, fairly common in pigs, not uncommon in dogs, but rare in sheep and goats. It is common in birds, particularly in the northern hemisphere, and also affects wild animals in captivity.

Tuberculosis in Domestic Animals and Other Animals.

Cattle.

Pathologically the disease in cattle has been characterized mainly by the formation of "pearls" or "grapes" on the serous membranes, and at one time it was quite common to refer to the bovine type of the bacillus as the *Perlsucht* bacillus. Although the nature of the lesion differs in some respects from that in man, there is the same basic difference in the reaction to infection in virgin soil from that in tissues sensitized by a previous or existing infection. In cattle the reaction in the regional lymphatic glands overshadows the reaction in the primary focus, which may even be wholly absent. Chronic tuberculosis associated with endogenous or exogenous reinfection is characterized by the freedom of the regional lymphatic glands from tuberculous changes. Early calcification, the relative absence of softening and cavitation, and the massive granulations on the serous membranes are all characteristic of bovine tuberculosis. The frequency of mammary tuberculosis, which may occur in from 2% to 4% of tuberculous milch cows, is in contrast with the rarity of the lesion in the human subject, and is of importance as a source of infection to pigs and human beings.

Infection of cattle is due in the vast majority of cases to the bovine type of bacillus. In some countries infection may occasionally be due to the avian type of bacillus; but the human type has not been recovered from tuberculous lesions in cattle.

Although the primary tuberculous process does not always continue to progress, complete healing is rarely, if ever, seen. As the main mass of information on tuberculosis in cattle is collected in the slaughter-house from the examination of young animals, and as most cattle found during life to be tuberculous are killed without delay, little opportunity exists of determining if recovery ever occurs. There is no evidence to suggest that the wide incidence of infection followed by complete recovery in a large proportion of cases in the human subject has any counterpart in cattle or other species of the lower animals.

[1] Read at the plenary meeting of the Fifth Session of the Australasian Medical Congress (British Medical Association), Adelaide, August, 1937.

Pigs.

Tuberculosis in pigs is most frequently due to infection by the bovine type of bacillus. In the United States of America and in Europe the disease is frequently due to infection by the avian type and sometimes by the human type of the bacillus.

The disease is usually produced by the ingestion of the milk of tuberculous cows or of infected food. Primary lesions therefore are found associated with the alimentary canal, and most frequently in the tonsils and the retropharyngeal, submaxillary and cervical lymphatic glands. The disease may also be localized in the mesenteric lymphatic glands. As the majority of pigs are slaughtered at an early age, the disease, when present, is usually localized. When the disease becomes generalized, the lungs are almost invariably affected, as are the liver and spleen. Tuberculosis of the bones is comparatively common, especially of the vertebræ, the ribs and the long bones.

Dogs and Cats.

Tuberculosis in dogs is most frequently caused by infection with the human bacillus, although the bovine type of bacillus is not infrequently the cause. The disease frequently presents the naked eye appearances of a disseminated neoplasm, with relatively large umbilicated tumours in the lungs, liver and kidneys. The primary lesions may occur either in the lungs or in the digestive tract. When generalization occurs the pleuræ and peritoneum usually become involved, and not infrequently the pericardium. Histologically the tuberculous lesion of the dog differs from that found in most other mammals. The characteristic epithelioid cell reaction, giant-cell formation and lymphocyte accumulation are usually absent. There is early and extensive coagulation necrosis.

The disease is similar in cats, but appears to occur less frequently than in dogs. It is most frequently caused by the drinking of milk from tuberculous cows, and infection is by the alimentary route. The mesenteric lymph glands are involved.

Sheep and Goats.

The disease is rare in sheep and goats. It has been found to be due either to the bovine or to the avian type of bacillus, and usually runs a chronic course.

The Horse.

Tuberculosis is not common in the horse. It is most frequently abdominal and is characterized by the development of tumour-like lesions in the mesenteric glands and spleen, and sometimes in the liver. These tumour masses often develop to a very large size. Primary thoracic tuberculosis also occurs, but is rare. In the majority of cases infection is by the bovine type of bacillus. From many of the cases investigated bovine bacilli of lower virulence than standard strains have been isolated. Griffith (1928) concludes that bovine bacilli become modified in their pathogenic properties during the progress of the disease, which is slow in the horse. This is of interest, for it is unusual for any of the three types of tubercle bacilli from warm-blooded animals to become modified by passage through a host of another species.

The Ferret.

The ferret is highly susceptible to the bovine type of bacillus. Infection results from ingestion of milk from tuberculous cows. The lesions are characterized by their content of enormous numbers of bacilli. Experimentally the animal is relatively insusceptible to the human type of bacillus.

Birds.

Tuberculosis due to the avian type of bacillus is fairly common in birds in most countries, but is comparatively uncommon in Australia. Gallinaceous birds are the most frequently attacked. The disease may be found in parrots. Canaries, finches and sparrows are very susceptible. All domestic birds, such as fowls, pigeons, pheasants, guinea-fowl and peacocks, are liable to infection; but turkeys, ducks and geese appear to be more resistant. Infection usually occurs through the alimentary canal. The intestinal tubercles are situated mainly in the submucosa. Ulcerative lesions also occur. The liver is almost invariably involved and the spleen very frequently. The lungs are involved in less than half the cases, while the bones and joints are affected in about 25% of cases. In the larger lesions there is well-developed caseation; but calcification is uncommon. Acid-fast bacilli are present in great numbers in the lesions.

Parrots and canaries are susceptible to both avian and human types of the bacillus. Infection by the human type is due to the frequent close association of these birds with the human subject. Cutaneous lesions are common in parrots affected by the disease.

Tuberculosis in Free-Living Wild Animals.

It seems most unlikely that tuberculosis exists in wild animals completely out of touch with man and the domestic animals. It has been shown, however, by Paine and Martinaglia (1929) that the disease may spread among wild ruminants living under natural conditions in South Africa. Further, the disease has been found in crows shot on the wing in Canada by Mitchell (1929). Each of these incidents demonstrates the capacity of the disease to spread amongst animals in the wild state; but the spread has been from the domestic to the wild animals.

Tuberculosis in Wild Animals in Captivity.

The disease has been found in mammals, birds and reptiles in zoological collections in various parts of the world. It closely resembles the disease in the corresponding orders of domestic animals, except that the course may be more rapid. Fox (1925) and Scott (1930) have published the results of their experiences in the Zoological Gardens of Philadelphia and London respectively. It is unnecessary here to do more than mention the occurrence of the disease in captive wild animals

in order to demonstrate the association of the disease with civilization.

In reptiles there are caseo-purulent lesions, in which numerous acid-fast bacilli are to be seen. These organisms appear to be distinct from the tubercle bacilli of warm-blooded animals.

The Diagnosis of Tuberculosis in Animals.

Clinical Examination.

Early diagnosis of the disease in animals by clinical examination is usually impossible. In the later stages it may be possible. Physical methods of examination follow much the same lines as in human medicine. Collection of sputum for examination is not simple, as animals do not expectorate; but as they swallow their sputum it can be collected in a small bucket-like vessel passed into the œsophagus. Direct collection of the sputum on a swab passed down the trachea is also used in Europe.

Pleural and abdominal exudates can be collected for examination. However, none of these methods is relied on for diagnosis, as almost invariably when the disease is suspected the animal is subjected to a tuberculin test.

The Tuberculin Test.

A tuberculin reaction means that the animal is infected by the tubercle bacillus. An infected animal is either destroyed or placed in isolation. Treatment is never attempted, and it is doubtful if recovery ever takes place in the lower animal. The tuberculin test, therefore, has come to have the greatest value in diagnosis of tuberculosis in animals. Whatever method of conducting the test is used, the results are specific.

The thermal reaction following a subcutaneous dose of old tuberculin has been used extensively for diagnostic purposes, and until more recently was the method most commonly employed. This subcutaneous method, as it is usually called, involves the recording of pre-inoculation and post-inoculation temperatures of the animal, and absorbs much time. Much of this time can be saved by using the intradermal method, which, largely in consequence of this saving of time, is the method mainly employed in systematic testing of herds of cattle.

Old tuberculin is probably the most reliable product for use in the subcutaneous test; but newer types of tuberculin, produced by the growth of the bacillus on synthetic media or other purified products appear to give more reliable results in the intradermal method. A double intradermal method, involving a second injection of tuberculin into the same site as the first if no reaction appears within from forty-two to seventy-two hours, has been introduced by British workers and has found much favour.

It would appear that the specific substances in tuberculin responsible for the thermal reaction and the lethal effect on infected guinea-pigs are of lower molecular size than those responsible for the reaction following intradermal inoculation.

In the early stages of infection sensitization may not be sufficiently strong to enable a tuberculin reaction to be obtained, and in very advanced tuberculosis of long standing it may be impossible to obtain a reaction.

The tuberculin test has not the same practical value in human medicine; but the absence of a positive reaction must be of great value in some cases.

The Control of Tuberculosis in Animals.

Tuberculin Testing and the Destruction of Infected Animals.

Systematic tuberculin testing and destruction of infected animals are an ideal method for the eradication of tuberculosis from herds of cattle, particularly when the incidence is not very high. The method is costly and is not usually economically possible, although it has been employed in the United States of America, where Congress makes appropriations of large sums for the purpose. Great progress has been made in those States in which the incidence was low. It is necessary to repeat the test at intervals, as some animals in the early stages of infection will be missed. The method is used in most countries for the establishment of accredited herds, and is economically sound for stud and other valuable stock.

Other systems have been introduced in an attempt to deal with the disease more economically. The Bang system aimed at gradually eliminating the disease by removing the calf to a clean environment immediately after birth. Universal adoption of the system has been impossible on account of its inherent intricacies. Another system aims at the removal and destruction of all animals that are contagious, thus protecting the public and gradually reducing the incidence of the disease in the stock. Such a system can be successful only in proportion to the efficiency of the inspection. This is usually inadequate, with the result that, in spite of some improvement, the disease never comes under complete control.

Tuberculosis in pigs depends upon transmission of the disease from cattle or fowls. If there are proper methods of animal management involving the sterilization of milk and the isolation of fowls in their own domain, the disease can be effectively controlled.

The eradication of tuberculosis of animals could be accomplished if we could afford to meet the cost.

Vaccination.

We have been forced by economic necessity to seek other ways of controlling and eradicating tuberculosis. The difficulties of increasing the resistance of animals to the tubercle bacillus have been recognized for many years. Under natural conditions recovery from a slight infection does not give rise to a lasting degree of resistance in man, while in the lower animal recovery does not occur or, if it does, is so rare as to be negligible. Koch observed that an infected animal would react differently from a normal animal to an injection

of bacilli. As in many infective diseases, there appears to be a "resistance of infection". This is particularly well manifest in syphilis and in diseases due to protozoan blood parasites. Protective methods have been developed on this basis, and the term "premunition" has been used to distinguish the state of resistance produced from that produced by true immunization.*

Calmette preached for many years that resistance to tuberculosis depended upon the presence of live tubercle bacilli in the tissues of the host. With Guérin he worked on the production of a non-virulent tubercle bacillus, and eventually one was obtained and given the name *Bacille Calmette-Guérin* or, more shortly, *B.C.G.* A considerable amount of work has been done on *B.C.G.* vaccination, first in an attempt to demonstrate if the culture is quite harmless to animals and man, and secondly to determine if ingestion or parenteral injection of the organism leads to a state of increased resistance or immunity to natural or artificial infection by virulent strains. At first a few workers threw some doubt on the permanent avirulence of the B.C.G. culture; but subsequent work has confirmed the opinion of Calmette and Guérin that the culture is innocuous.

A. S. Griffith (1931) carried out a series of tests on monkeys, selecting them for experiment on account of their high susceptibility to tuberculosis. He found that the *B.C.G.* culture was incapable of giving rise to progressive tuberculosis in the rhesus monkey, although the organisms might remain alive in the lymphatic glands for periods up to seven months, provoking no characteristic tuberculous lesion. He found further that by oral administration, larger doses given over longer periods than those recommended by Calmette were necessary before the organisms passed in any numbers through the mucous membrane into the adjacent lymphatic glands. In his experiments vaccination with *B.C.G.*, either by feeding or by injection, failed to give to monkeys the complete protection previously reported by Wilbert, but in some instances might have produced a low grade of relative immunity.

The most extensive trial on animals is probably that carried out by Watson (1934) on cattle in Canada. For the purpose of the observations, new-born calves in a tuberculous herd of dairy cattle were vaccinated. The vaccinations were repeated yearly. The observations were extended over a period of ten years. Fifty-four vaccinated animals and thirty-six unvaccinated controls came to necropsy at various periods during the trial. At no age was there found to be a significant difference in the incidence of the disease between members of the two groups. In the vaccinated animals, up to two and a half years of age, the disease, when present, was slightly less extensive than in similar animals of the control group. In the 90 animals examined at all ages up to five and a half years the aggregate incidence of the disease was the same in each group, 83·4%. There was no evidence, therefore, that the vaccination protected the animals against natural infection, although there was slight evidence of some modification of the tuberculous process in the first two years of life.

Buxton (1934) reported the results obtained by himself and A. S. Griffith and Glover in Great Britain. The *B.C.G.* culture was found to be avirulent for calves. Intravenous injection was found to be the most satisfactory method of vaccination. The degree of increased resistance varied directly with the dose. The most satisfactory dose was found to be 10 milligrammes, followed three weeks later by 100 milligrammes. This double intravenous inoculation gave complete immunity three months later to cultures of virulent bovine tubercle bacilli administered orally. There was a progressive decrease in resistance with lapse of time, until at the end of twelve months it had almost completely disappeared.

The evidence seems to show that oral administration of *B.C.G.* to animals is useless unless large and repeated doses are given. Under these conditions the degree of resistance produced cannot be expected to be as good as that produced by subcutaneous or intravenous administration. Subcutaneous and intravenous administration appears to give some temporary increase in resistance. According to the evidence, this increased resistance cannot be strengthened or even maintained by repeating the subcutaneous inoculations at yearly intervals. Intravenous inoculations appear to establish a somewhat higher degree of resistance. Evidence is not yet available on the effect of repeated intravenous injections over a period of years.

The results obtained by the use of *B.C.G.* do not encourage us to believe that the method will be found to possess any value in the control of bovine tuberculosis. Experiments in Australia have given no more encouraging results than those obtained in other countries.

The experiments carried out with the Spahlinger vaccine on cattle in northern Ireland were slightly encouraging, but were inadequate and gave no definite and conclusive results.

Vaccination will probably never prove an adequate solution of the problem of the eradication of bovine tuberculosis.

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THE PATHOGENESIS AND PROPHYLAXIS OF PULMONARY TUBERCULOSIS.[1]

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MY object this morning is to offer you a concise expression of modern thought and teaching on the pathogenesis and prophylaxis of chronic pulmonary tuberculosis.

Pathogenesis.

The following scheme of "pathogenesis", which I shall now elaborate, is intended to serve as a foundation on which to build a working scheme for an intelligent and successful attack on the more important practical problem of prevention.

A. Infection:
  (*a*) Childhood.
  (*b*) Later life.
B. Reinfection:
  (*a*) Endogenous.
  (*b*) Exogenous.
C. Pre-clinical disease discovered:
  (*a*) By survey—(i) milieu, (ii) group, (iii) community.
  (*b*) In retrospect—abortive tuberculosis.
D. Manifest disease.

The evolution of tuberculosis is dependent on two factors, infection with the tubercle bacillus and the resistance, both natural and acquired, of the individual. That particular form of the disease, and the most common, known as chronic pulmonary tuberculosis (often called incorrectly the adult form of pulmonary tuberculosis or, more simply, phthisis) may be defined as a disease which has developed in a person who has been previously infected by and who has thus attained a specific increased resistance to tubercle bacilli, or, as Dr. Bull has called it, premunition. I thank him for the word. It is forty-five years since Koch demonstrated that a second infection is necessary to produce a chronic progressive tuberculous process, but this axiom is not always appreciated. Distinction must be clear-cut between infection and disease; failure to appreciate this point leads to confusion of thought and to arguing at cross purposes.

Most individuals within the bounds of civilization are infected with the tubercle bacillus at some time during their lives, but in the vast majority disease does not follow. In a certain percentage, however, disease develops at a later period, owing to reinfection either from within the body or from outside, and associated usually with lowered resistance on the part of the individual.

The problem of infection in pulmonary tuberculosis has received special attention recently. For many years it was accepted that infection in civilization is universal in childhood, and that phthisis can arise only from the recrudescence of this childhood infection, that is to say, by endogenous reinfection. It was further accepted as a corollary that no civilized adult could be harmed by external contact with the tubercle bacillus; in other words, that he was immune to all but his own tubercle bacilli. This doctrine is opposed to reason and experience, and it is being abandoned.

American workers, however, in their zeal to prove its falsity, have gone to the other extreme. Opie and his co-workers,[1][2] of the Henry Phipps Institute in Philadelphia, have attempted to prove that adult tuberculosis is not a lighting-up of a childhood infection, but is acquired by primary infection during adolescence or even adult life. A perusal of American literature tends to give the impression, put into words by a recent contributor[3] to THE MEDICAL JOURNAL OF AUSTRALIA, that tuberculosis is "a contagious disease like many of the acute infections".

There is need at the moment for moderation in thought and word on this subject. That there is no tuberculosis without the tubercle bacillus is a truism; but it has required careful study for many years to prove that actual tuberculous disease occurs more frequently among those who have lived in a tuberculous environment than among those who have not. It has been pointed out, indeed, that no one knows yet whether more cases of pulmonary tuberculosis would be found among 10,000 "contacts" than among 10,000 persons living in abject poverty.[4] It has never been necessary to publish careful statistical surveys in order to prove the infectivity of scarlet fever or diphtheria; the fact is obvious. There are strong arguments against attaching supreme importance to infection in the development of pulmonary tuberculosis, as for instance the fact that many persons live in close contact with tuberculous patients for years without contracting the disease. Thus resistance rather than infection may be the dominating factor in deciding the ultimate story. Burrell[5] expresses concisely British opinion on this controversial subject, and I commend his words to you:

> Let us agree that the individual, child or adult, is not likely to catch consumption from a patient, provided there is reasonable cleanliness and ventilation; let us agree that a mild infection does confer some slight but variable protection; but to deny the infectivity of tuberculosis to the adult as well as the child is a dangerous doctrine.

It is agreed by all that primary infection occurs most frequently from contact in the home with a person suffering from the disease. It is further agreed, however, as the result of recent surveys, that large numbers of children are now growing to adult life without sustaining this primary infection; and there is an increasing number of persons in any community who fail to react to the tuberculin test. This test, as described by Mantoux, consists in the intradermal injection of a measured amount of tuberculin. A wheal occurring in some forty-

[1] Read at the plenary meeting of the Fifth Session of the Australasian Medical Congress (British Medical Association), Adelaide, August, 1937.

eight hours at the site of injection is regarded as a positive result, indicating that the individual has been infected by the tubercle bacillus and is sensitized to its proteins. The scratch test of Pirquet is less sensitive, but when its result is positive is of identical significance.

To illustrate my remarks, I should like to show you some results of the Mantoux test as carried out at the Royal North Shore Hospital, which have been tabulated by my colleague, Dr. Douglas Anderson (Table I). You will notice that they show lower percentages of positive reactors, especially among "contacts", than are found in large cities abroad. The figures are based on 875 tests made on non-tuberculous persons. In 701 "contacts" a positive result was obtained in 67%. Among 174 "non-contacts", that is to say, patients in whom there was no history of contact with any tuberculous person, only 38% gave a positive reaction. It must be stated that we have been obtaining a higher percentage of positive reactions during the past eighteen months, since using for the test the special tuberculin "P.P.D." of Parke, Davis and Company.

TABLE I.

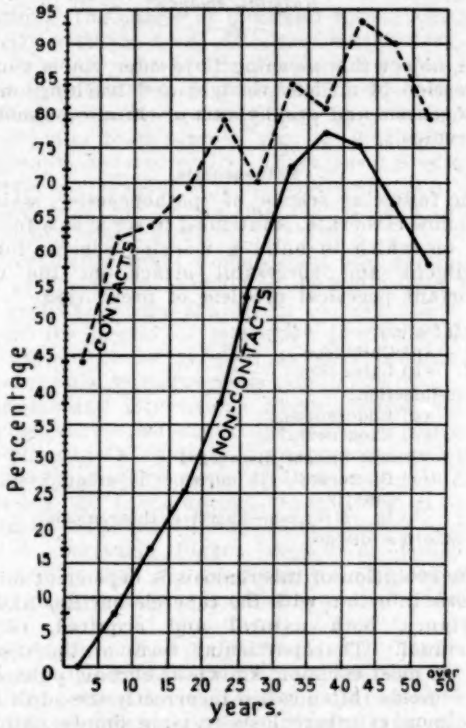
Royal North Shore Hospital Pulmonary Clinic.

Mantoux Tests in Non-Tuberculous, 1931 to 1937.

Ages.	Non-Contacts.			Contacts.		
	Number Tested.	Number of Reactions.	Percentage.	Number Tested.	Number of Reactions.	Percentage.
1 to 5 ..	15	0	0	109	48	44
6 to 10 ..	36	3	8	138	85	62
11 to 15 ..	18	3	17	123	77	63
16 to 20 ..	12	3	25	64	44	69
21 to 25 ..	13	5	38	47	37	79
26 to 30 ..	17	9	53	38	26	70
31 to 35 ..	18	13	72	45	38	86
36 to 40 ..	13	10	77	52	44	81
41 to 45 ..	8	6	75	35	33	94
46 to 50 ..	9	6	67	19	17	90
Over 50 ..	15	8	53	29	23	79
Totals ..	174	66	38	701	473	67

A consideration of the graph reveals some points of interest. First, infection in "non-contacts" is very slight in childhood, but it rises steadily to the fifth decade. This suggests that primary tuberculous infection is not infrequent in adults, a statement borne out by inspection of the "contact" figures. Secondly, you will note the much higher percentage of reactions among the contacts in every age group, but most significantly in the first fifteen years of life. These figures are a striking illustration of the importance of home contact in the dissemination of tuberculous infection.

Once infection has occurred, provided it does not overwhelm the individual, he acquires immunity, as well as that condition of sensitivity to tuberculoprotein which von Pirquet called allergy and which is demonstrable by cutaneous tests. Allergy and immunity are not identical, but their complex relations cannot be discussed here. I would repeat that in the vast majority of persons first infection gives a lifelong immunity.



Reinfection, which may happen at any time from within a few months to many years after the initial infection (a variable interval, by the way, that is not always appreciated) can occur in two ways: first, as I have already mentioned, by reactivation and dissemination of bacilli already present in the patient's own body, that is to say, by endogenous reinfection; secondly, by further bacilli introduced from without, that is, by exogenous superinfection. I am still of the school which believes that in the majority of cases reinfection is endogenous. We know that patients who are apparently cured may relapse after many years, twenty-five or even fifty, and go rapidly downhill. "Never say a patient is cured of pulmonary tuberculosis until he is safely dead of some other disease" is Wingfield's aphorism. Also we find often enough tuberculous metastasis to joint, bowel, testis, and so on. It is unnecessary to suppose, bearing in mind evidence of the viability of tubercle bacilli found *post mortem* in old healed lesions, that a patient suffering either from relapse or metastasis has been reinfected from without.

The evidence in favour of endogenous reinfection is unassailable, but I do admit that exogenous

reinfection occurs as well and that it may be a source of danger. The spread of active pulmonary disease through several members of a family can be readily and adequately explained when we know that there has been ample opportunity for intimate contact and repeated dosage. It would be a folly no medical man should countenance to allow the Mantoux-positive, that is to say, the already infected members of a family, whether children or adults, to associate with the tuberculous sufferer without taking any precautions whatever.

Broadly speaking, the two main factors in determining the outcome after reinfection, as in infection, are the size of the dose of bacilli (and perhaps their virulence) and the resistance of the individual. Obviously, with a massive dose disease is more likely to occur, though repeated small doses may eventually produce the same result.

The subject of resistance is a complex one, and I can only touch on it. It is extraordinary how persistently the public clings to the belief that tuberculosis can be inherited, despite the entire absence of any scientific evidence in its favour. An infant cannot be born with tuberculosis, but there is no doubt that in some families there is a specific constitutional weakness or diathesis that renders them more vulnerable than the average to the tubercle bacillus. Resistance, or the lack of it, may be inborn, and there is more than a grain of truth in the old teaching that phthisis occurs only in those who are predisposed to it. Resistance may also be racial, as is evidenced by the comparative immunity of the Jewish people, and by the susceptibility of the Irish and of most of the dark races.

More important than natural susceptibility is the breakdown of individual or occasionally, as occurred during the War, communal resistance. It is a significant fact that the importance of tuberculosis varies inversely as the social and economic status of the community. Poverty is the handmaiden of consumption, bringing in its train overcrowding with lack of sunlight and ventilation, inadequate nutrition, overwork, lack of sleep, and especially mental anxiety and worry. The influence of certain occupations must be remembered, notably those in which silica is involved. Alcohol and injury may determine the onset of disease, and pregnancy seems at times to be a factor.

Reinfection, then, may occur often and be innocuous, but if the dose is large enough or the soil is sufficiently receptive, then disease results. In its inception it often gives no indication of its presence to the person concerned. Herein we have a modern medical paradox, that in a person with chronic pulmonary tuberculosis the diagnosis should be made before he is aware that anything is amiss. Of course, this applies to many other diseases, though not, I think, with equal force. The recognition of subclinical disease, namely pulmonary tuberculosis which is active and progressive, but which causes no symptoms whatever, is due entirely to radiography. Some clinicians may still be sceptical as to whether X rays can reveal active disease if the patient lacks symptoms; if such there be, I urge them to read the recent literature, and I shall show skiagrams of one patient which illustrate radiologically the spread of disease, in the absence of signs or symptoms. Such patients are not uncommon, and no doubt you have all seen them. Whether the evidence of a single skiagram can be accepted as sole proof of active disease is open to doubt, but serial X ray photographs can certainly show progressing disease in an individual apparently perfectly well.

The importance of this latent group is to be stressed. Wingfield and MacPherson,[6] after surveying radiologically 2,400 workers, state that probably 1% of an adolescent working class population have latent pulmonary tuberculosis, and that from these arise the clinically tuberculous patients. They emphasize the fact that pulmonary tuberculosis during long periods of its activity is often a symptomless disease. These cases can be found only by systematic radiological survey, to which I shall refer later.

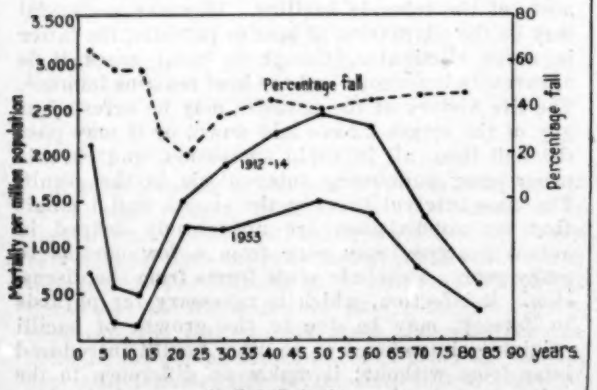
Subclinical disease does not always progress to manifest disease. The discovery in the lung, radiologically in later life or *post mortem*, of healed tuberculous areas of considerable extent, of which the patient showed no signs, is proof thereof, but this knowledge gives us no warrant to ignore latent pulmonary tuberculosis when we find it, particularly in the adolescent. It is our duty to discover, if possible, all patients with latent disease, and to observe them carefully in an endeavour to determine whether the disease is active or healed. Unless there is convincing evidence that the lesion is healed when it is first discovered, it is probable that manifest disease will follow sooner or later.

Let us summarize the position. Infection, which may occur at any age, is due to direct or indirect contact with a person whose sputum contains tubercle bacilli. Most of us at some time in our lives, especially if we are city-dwellers, become hosts of the tubercle bacillus. However successful may be the adaptation of host to parasite, the latter is never eliminated, though in most cases it is apparently innocuous and the host remains immune. The life history of the parasite may be arrested at any of the stages I have laid down, or it may pass through them all in rapid succession, and chronic progressive pulmonary tuberculosis is the result. The time interval between the stages, and I admit that my subdivisions are not clearly defined in actual practice, may vary from a few months to many years. I exclude acute forms from the discussion. Reinfection, which is necessary for phthisis to develop, may be due to the growth of bacilli originally introduced or to other bacilli introduced later from without; it makes no difference to the subsequent development of the disease. It is clear that with subminimal doses only microscopic lesions may result. No one can say how many people remain at Stage B throughout life; nor do we know, in the majority of cases, at what point in time a

person has been reinfected; but this is not of practical moment. Sooner or later, in a certain percentage of "hosts", immunity fails and reinfection makes inroads on pulmonary tissues which are macroscopic; and what is more significant, they become radiologically apparent.

Pre-clinical, latent or asymptomatic disease, call it what you will; the term has come to stay, and the recognition of this group may be considered a milestone in preventive medicine. How often this latent disease remains as the end-result in the individual is yet unknown, and years of continuous careful surveys must elapse before this question can be answered. In a small minority of cases the bacillus pursues an inexorable course to the time when its impact makes the host finally aware of its presence, and the physician is then as a rule faced with one of the most difficult problems in the whole of medicine, namely, the curing of a patient with pulmonary tuberculosis at an advanced stage. This small minority makes an impressive showing in world statistics of mortality, as you will agree when reflecting on the fact that among young people between the ages of fifteen and twenty-five years the death rate from pulmonary tuberculosis is more than half that of all other diseases taken together.

I should like to draw your attention here to two graphs which show the heavy toll paid by young adults. They show the contrasted mortality from tuberculosis in England and Wales between the years 1912 to 1914 and 1933, for both sexes, with the percentage fall at each age period. They are similar graphs to those shown by Dr. Holmes. You will notice that the mortality has fallen for each period, but that the fall has been greatest in childhood, while it has been slight at the ages of twenty to twenty-five years, especially for females. Two conclusions may be drawn: (i) exposure to risk of infection has been much reduced in childhood; (ii) life is being lived too strenuously by young women at the present day.



Prophylaxis.

The following summary of the prevention of pulmonary tuberculosis was drawn up before D'Arcy Hart's Milroy Lectures[7] for 1937 appeared in *The Lancet*. If any of you have not read them, I strongly advise you to do so, as they present in careful and convincing array, and far more fully than I can hope to do, the modern argument of this problem of preventive medicine. Also it is inevitable that I should cover in part the same ground as Dr. Holmes; all that he has said I endorse wholeheartedly.

Three objectives:

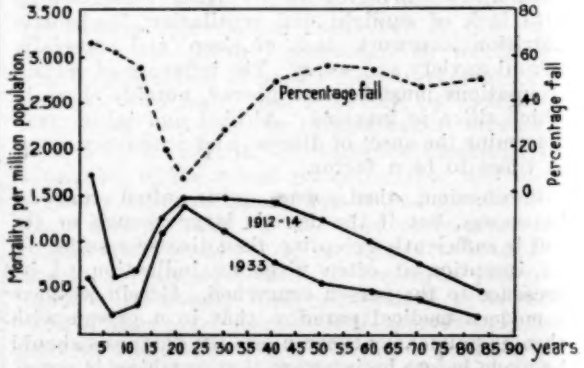
1. To prevent infection.
2. To prevent reinfection.
3. To prevent latent disease becoming manifest.

Four main lines of attack:

1. Control of open cases.
2. Systematic examination of contacts.
3. Community surveys.
4. Increasing resistance, racial and individual.

For any campaign to be successful there are four preliminary essentials: an adequate public health organization, a recognition of the part played by the general practitioner, a careful education of the layman, and last, but not least, a thorough education of the medical student in chest diseases, especially tuberculosis. Assuming these to be efficient, I shall proceed to discuss the scheme I have given you.

The three objectives I postulate are based, of course, on my scheme of pathogenesis, as are the methods of attack presented in my summary. At the outset it may be asked: Can we prevent primary infection by what has been called a ubiquitous and unexterminable germ, or should we really try? I have no doubt that we should try as far as possible to prevent infection, not only in the child, but in the adult. If the infection be very large (the "massive dose") the patient dies rapidly, and this happens all too frequently in childhood. And even though this first infection may be harmless at the time, and may confer a relative immunity, we must not forget that it sets the stage for all the subsequent developments.



The greatest source of danger is the sputum-positive patient, and a recognition of this fact brings up a problem that has two angles, those of clinical and of preventive medicine.

The clinician must consider the welfare of his patient, and his views may clash with those of the

public health official who is concerned with the community at large; but this problem can in most cases be solved by that cooperation which is essential to the success of any scheme. "The removal of the source of active infection from the family is of fundamental importance", stated the National Health and Medical Research Council at its meeting at Hobart in February of this year, and with this we all agree, at least in theory.

Perhaps legislation may be required to ensure segregation, but this is a moot point. What is quite certain is that there must be adequate accommodation available for all, especially for advanced patients. I would also press for the establishment of more hospital beds for the treatment of the earlier patients, laying stress on the need for complete and prolonged rest and for more aggressive treatment than is permissible in the usually understaffed sanatorium. There is need for reform in what is complacently called "sanatorium treatment" in this country; it has in many instances fallen far below modern standards. And I would emphasize the value of collapse therapy as a means of preventing the dissemination of tuberculous sputum.

If we succeed in our first objective, the battle is won; but this will not be for many years, if ever. I shall proceed to the second.

Infected persons are discovered by the tuberculin tests in the systematic examination of "contacts" and in group surveys. At this stage (if they have not been removed before infection occurred) it is very desirable in the tuberculous household that either the patient or the children should be removed from the home, in order, so to speak, to "break" contact. If the patient cannot be isolated, care in the home can minimize the risks, whether of infection or of exogenous reinfection, but much more should be done in caring for and isolating infected children. I agree with Dr. Holmes that country preventoria should be provided on a large scale, where such children might stay for long periods, and certainly until such time as the source of infection has been removed from the home. It is futile for the child to return to a tuberculous environment, to be exposed to further infection. It might be advisable to adopt a system of boarding out these children, in view of the success of the Grancher system in France. Meanwhile from this group hundreds of cases of chronic pulmonary tuberculosis are recruited annually in this country.

I have pointed out that a long latent period may occur between infection and disease. It is probable that the average interval in adolescents is about two years, though it may be twenty or more when young children are infected, and this fact has an important practical application, which may be noted here. The contacts are usually willing to be examined, especially when goaded by fear after the death of the patient. A single examination at this time, however thorough, may do irreparable harm if the "contact" is assured that all is well and is not warned to report at regular intervals. I have seen distressing examples of this, when the patient, living in a fool's paradise, ignored symptoms which otherwise could not have failed to lead to medical examination.

Examination of contacts is quite simple, and it can be carried out inexpensively. It should be adequate; that is to say, it should include all persons, especially children, with whom the patient has been in close contact. The father and mother must be examined; sometimes this reveals the infecting source, to the general surprise. Nor should older people escape examination; grandfather's so-called "bronchitis" may have infected many grandchildren.

At the Royal North Shore Hospital we perform the Mantoux test on all "contacts", using the tuberculin "P.P.D." of Parke, Davis and Company; Dr. Bull has mentioned the specific desirable fraction. This, or a similar preparation, is now being accepted as a standard test medium throughout the world, and it has superseded old tuberculin. If the "contacts" do not react, their chests are not examined, but they are ordered to report again in six months. They attend at regular intervals, and school-children always come in their holidays. If contact has not been broken, they are subjected to the Mantoux test regularly, but not unless or until they give a positive reaction are their chests examined by X rays. This routine saves both time for the physician and money for the hospital. I would emphasize the great importance of the intradermal tuberculin test in effective tuberculosis control; if there is a positive result, it is proof of infection; if there is no result, infection, with few exceptions (notably after acute fevers), has not occurred. And I would repeat that infection is no longer universal in childhood. Chadwick[8] analysed the result the result of testing 400,000 children of school age in Massachusetts with the von Pirquet test, and found that only 25% gave a positive reaction.

The positive reactors are then examined by X rays, and if there is radiological evidence of tuberculous disease endeavour is made by clinical examination and careful appraisement of history and symptoms to ascertain whether it is active or inactive. If there is any doubt the patient is always given the benefit; that is to say, he is sent to a sanatorium. If it seems reasonable to believe that the disease may be inactive, he is kept under close observation and serial X ray photographs are taken at appropriate intervals of a few months. Each case, however, is judged on its merits, and special note is taken of the economic and domestic background.

Interpreting "prevention" in a broad sense, early diagnosis is a primary line of attack. It seems scarcely credible that in England in only 12% of patients is a diagnosis made at an early stage, and I believe that we can show better figures in Australia. But in the majority of cases the diagnosis is first made when the disease is in an advanced stage. The medical practitioners can and should do more to recognize the early stages, and

he can no longer be excused for the omission of a skiagram in a doubtful case, especially when no chest signs can be found. How often do we hear the statement: "I thought of having an X ray photograph taken, but I could find no physical signs." In pulmonary tuberculosis X ray signs have utterly routed physical signs. General recognition of this fact will lead towards earlier diagnosis, but I admit that it will not bring the patients any earlier to the doctor. As Dr. Holmes has pointed out, the working man cannot afford to be "an early case of pulmonary tuberculosis"; Dr. Holmes has suggested a solution to this problem. By a carefully controlled publicity campaign a small percentage of patients will be induced to present themselves earlier than hitherto, as soon as suggestive symptoms appear; but this will not suffice for our purpose.

It is necessary to tackle the problem anew, from an entirely different angle. And here I cannot do better than quote to you Dr. D'Arcy Hart:

> For achieving early diagnosis in the past we have tended to concentrate upon the early clinical case, i.e., the patient who already has symptoms or physical signs. In the future we must concentrate upon the preclinical case. In the past we have for the most part attempted to recognize the presence of progressive lesions, soon after symptoms have developed, by a single examination at the request of the individual when he feels ill. In the future we must try as far as possible to discover these lesions before the onset of definite symptoms by the routine periodic examination of selected groups of individuals, particularly young adults, supposedly in health. Serial radiograms and (especially where a low tuberculous infection incidence prevails) Mantoux tests would be the basis of such examinations.

This may be considered by some a revolutionary doctrine, but I am convinced of its soundness. Periodic health examinations must be carried out, at first of selected groups, especially young adults, but later it may be practicable to survey whole communities, as has been done already in Scandinavia. The examination would be based primarily on radiography or perhaps fluoroscopy. With modern screens, fluoroscopic examination of the chest has become very accurate, and the percentage of error is quite small. Wingfield and MacPherson used screening in their survey of 2,400 adolescent workers, taking films only as a precaution in doubtful cases.

I believe that the X ray survey of selected groups will prove to be one of the most profitable advances in preventive medicine, provided, and this is an essential qualification, that the utmost care is taken in the interpretation of films. Objections will be raised, such as the cost, the creation of phthisiophobia, and the difficulty of persuading patients to accept the stigma of sanatorium treatment when they do not feel ill. Dr. D'Arcy Hart has met them all, and I refer you to his paper.

In my limited time I can only draw your attention, without comment, to the importance of the education of the patient and of general home management, and I can only mention briefly some methods of increasing resistance. "The economic factor is definitely the most important aspect of the campaign against tuberculosis"; this the National Health and Medical Research Council has laid down as its first consideration. I am not so sure of the truth of this as I once was, but I agree that social and economic factors are of tremendous importance. Overcrowding and under-nourishment are powerful allies of the tubercle bacillus. Therefore, the standard of living among the poorer people should be improved and an endeavour should be made to ensure an adequate diet for all, especially children, always remembering that "adequate diet" no longer means merely a diet containing a sufficient number of calories. It implies a balanced diet, with plentiful amounts of the protective substances, vitamins, minerals and fats, especially milk. Tuberculosis, it has been pointed out, "is the one disease to which there does seem to be fairly good evidence that a well-fed body is more resistant".[9] The raising of resistance, both individual and communal, is essential if we are to cope with the disease. Particularly, as I have previously pointed out, is resistance significant in determining whether reinfection will lead to progressive disease, though, of course, it has a bearing as well on the ultimate results of treatment.

Let me add one last word on *B.C.G.* vaccine, concerning which evidence is accumulating to show that it is at least of temporary value and that it gives partial protection not only in infants, but also in adults. It appears, for instance, to have been helpful in protecting "Mantoux-negative" nurses in Norway from acquiring the disease.[10] More work on this is required, however, and English-speaking countries still await proof from continental Europe of the value of Calmette's famous vaccine.

In conclusion I would plead for a more sympathetic and practical consideration by public health authorities and the various governments of the Commonwealth of the problem of tuberculosis control. Federal and State governments have had placed before them by competent authorities carefully considered programmes, but we still await the day when practical moves will be made. I state emphatically that in this respect in Australia we are lagging behind most countries, even admitting that our mortality is among the lowest in the world. As this is one of the most costly diseases to the State from an economic standpoint, to spend more money in combating it will ultimately prove an economy to the enlightened government that undertakes the expenditure. A continuance of the present policy of apathy and delay in financing a comprehensive campaign against tuberculosis, a preventible disease, which in Australia is responsible for an annual death rate of nearly 3,000, more than half of whom are under the age of forty-five years, is not merely a moral crime, it is an economic blunder.

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CHRONIC MYOCARDITIS.[1]

By R. WHISHAW, M.B., M.R.C.P.

MY object tonight is to make a plea for abandoning the term "chronic myocarditis" in clinical practice. I shall try to prove to you that in doing so you will confer benefit on your patients and on yourselves. "Myocarditis" means inflammation of the heart muscle; but this occurs only in rheumatic fever (when "carditis" is a better term), in diphtheria as an acute form only, and very rarely in typhoid fever and pyemia. It is a pathological entity and not a clinical one. Chronic myocarditis, however, has been used largely in the past as a scrap-heap for various conditions in which it was thought that some weakness or loss of reserve of the heart muscle existed.

During the past eighteen months I have investigated a number of patients, including many war pensioners, whose medical certificates, even recent ones, indicate that chronic myocarditis is still being used as a diagnosis. Hence this paper. The abandonment of the term in clinical medicine would lead to more accurate diagnosis and so to correct treatment, and would also tend to save many unfortunates with functional heart disease from chronic and incurable invalidism.

During the following discussion an attempt will be made to elucidate some points in the investigation of these cases; but my main theme, as already mentioned, is to urge that chronic myocarditis be abandoned as a clinical diagnosis. Let us first consider the type of case to which the term usually applies.

The common complaints are shortness of breath on exertion, palpitation, often with premature beats, and pains in the chest. These symptoms are common to functional disorders and organic heart disease, and quite frequently in the cases I have investigated a functional disorder was found, although it had been previously labelled "chronic myocarditis". The following is a case in point.

CASE I.—A male, aged thirty-six years, a returned soldier, complained that he had had pains in the chest and shortness of breath on exertion for eight years. The pain was in the region of the apex of the heart, was stabbing in character, and sometimes radiated to the left arm. It had no definite relation to exertion, lasted for hours, and might come on during rest. The chest often felt sore afterwards. He suffered occasionally from palpitation, and once had lost consciousness after such an attack.

On examination he was found to be obese, weighing 94·5 kilograms (fifteen stone). There was some dyspnœa on slight exertion; but his colour was good. The apex beat was not palpable. The heart sounds were regular and normal. The systolic blood pressure was 130 and the diastolic pressure 90 millimetres of mercury. The thyreoid was just palpable. The peripheral arteries were soft and the retinal vessels were normal in appearance. The heart rate when the patient was lying down was 80, and when he was standing 96 per minute.

On the employment of an exercise tolerance test the respiration rate rose quickly to 50 per minute, but soon subsided, and the pulse rate varied in proportion. The electrocardiograph was normal, apart from low voltage in the *Q R S* complex in Lead III and a negative (that is, inverted) *T* wave in Lead III. Fluoroscopic examination revealed a "horizontal" type of heart, due to a high diaphragm, but no enlargement. The aorta and lungs appeared to be normal. The conclusion was that there was no organic heart disease and that the dyspnœa was partly due to obesity. The diagnosis was "obesity and *angina innocens*" (see below).

On occasions the diagnosis of chronic myocarditis has been applied to the following: hypertensive heart disease, coronary disease, "masked" thyreotoxicosis, chronic bronchitis with emphysema (with or without *cor pulmonale*) and, more rarely, aortic stenosis, cardio-vascular syphilis, anæmia and obesity (Levine, White).

The pathological changes in the myocardium are not truly inflammatory, but are in the nature of degenerative fibrosis. There is a very small group, in which cardiac enlargement, ultimately ending in congestive failure, occurs without a satisfactory pathological explanation. This is best termed "chronic myocardial insufficiency (?) cause" (Levine). Before a diagnosis of heart disease is made it is useful to attempt to answer these four questions as a routine procedure: (i) Is there any evidence of organic heart disease? (ii) If so, what are the structural changes present? (iii) What are the functional changes? (iv) What is the ætiology?

Evidence of Organic Heart Disease.

Let us now analyse these four questions. After the routine careful history and complete examination it is often possible to answer the first. In many cases there will be doubt, and then further investigation must be carried out without delay, for the first consultation is always the most important. The heart is examined by fluoroscopy and an electrocardiograph is taken. Before this is done, however, the symptoms and signs have to be evaluated and, as already mentioned, perhaps the most common of these include the following: dyspnœa, palpitation, a systolic murmur and chest pains.

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on September 15, 1937.

Dyspnœa.

Dyspnœa is very common. The patient should be gently exercised to see if dyspnœa is really present and, if so, to what extent. Functional dyspnœa is frequently largely subjective, and complaints are made of difficulty in getting a really good breath, or of sighing. It must be borne in mind that poor response to exercise is not proof of organic heart disease, although it is an early and valuable sign. Functional dyspnœa complicates organic heart disease in about 10% of cases, and its importance is then rendered more difficult to assess.

The presence of dyspnœa having been established, causes other than cardiac must be eliminated. Emphysema is a common cause, and obesity still more common. Obesity leads to raising of the diaphragm and "squat" or horizontal position of the heart, with consequent interference of function. Primary or secondary anæmia, with hæmoglobin values below 65%, lack of exercise, and any condition which lowers the general tone are all sometimes responsible for dyspnœa.

Palpitation.

Palpitation, especially if accompanied by premature beats, often leads to the suspicion of heart disease. If premature beats occur despite a rapid heart rate, thyreotoxicosis should be suspected, for usually tachycardia leads to their disappearance. Persistent tachycardia in a child should make one think of rheumatic carditis. If the attacks of palpitation begin and end suddenly, paroxysmal tachycardia is suggested; but patients vary a great deal in what they mean by palpitation, and careful questioning will often reveal the fact that premature beats are responsible. Premature beats, in the absence of any other sign of heart disease, especially enlargement, can safely be ignored.

Systolic Murmurs.

A systolic murmur at the apex, when discovered as an isolated phenomenon, and when it is soft, is rarely of significance; but its presence necessitates increased care in the physical examination. Every effort should be made to locate the apex beat and so estimate the heart's size. Percussion is rarely of much help in this direction, in my opinion, unless the patient is thin.

Anæmia should be considered as a possible cause, and also relative insufficiency from cardiac enlargement. In rheumatic mitral disease the bruit is usually more harsh, particularly if the heart rate be made to accelerate. A functional systolic murmur often alters or disappears on change of posture.

Chest Pains.

Pain in the chest is an important symptom, worthy of close attention. Pain in the left side of the chest is far more often due to functional causes than organic heart disease, in my experience. The danger lies in the fact that in about 25% of the cases of angina of effort there are no organic signs, even when there is grave coronary disease. Close questioning, however, usually solves the problem, the important thing being to ascertain whether the pain has a quantitative relation to effort.

A most useful clinical conception of cardiac pain is that described by Geoffrey Bourne in *The British Medical Journal* of April 3 of this year. He divides cardiac pain into four groups, as follows: (i) the pain of coronary occlusion, (ii) angina of effort, (iii) spasmodic angina, (iv) *angina innocens*. And I may add the pain of aortitis and aneurysm of the aorta.

The symptoms and signs of coronary occlusion are well known to you. In angina of effort the important point is the quantitative relation to exertion. But it must be remembered that the pain may come on with less effort after a meal or exposure to cold. It is of short duration, relieved by rest, situated substernally, and usually described as a squeezing type of pain, often with radiation to the neck and arm. The third type, spasmodic angina, is rare and need not detain us, as the cause is still open to doubt. *Angina innocens* is quite common in my experience. The pain is usually felt in the region of the left breast and has no definite relation to exertion, often occurring after the effort ceases; it is usually of long duration and stabbing in character, or dull and aching, and may radiate to the left arm and sometimes leaves a feeling of tenderness in the left side of the chest. Other symptoms are present, especially exhaustion, and sometimes shortness of breath, faintness, palpitation and giddiness. Respiration is often irregular and sighing, and the dyspnœa when analysed may appear to be more subjective than objective. This symptom complex is frequently, but not invariably, accompanied by neuro-circulatory asthenia or an anxiety state, and syncopal attacks are not uncommon. When the patient stands the pulse is often observed to increase in rate and the blood pressure to fall. Clinical examination, electrocardiography and screening reveal no organic heart disease. Some recent observers (Shirley Smith *et alii*) have found that the blood sugar content may be low.

CASE II.—A male, aged forty-three years, a returned soldier, complained of "awful" pains in his chest, mostly on the left side. He had had these symptoms for two years. The pains were unrelated to exertion and often occurred at night. He slept badly and had "dizzy turns" and headaches. His heart often thumped hard, and he was short-winded. He had had trench fever during the Great War and had been gassed, but not severely.

On examination the colour and nutrition were found to be satisfactory. The thyreoid was not enlarged; the skin was a little damp, but there was no tremor. The pulse was regular and its rate was 100 per minute at rest. The apex beat was palpable in the fifth intercostal space 8·75 centimetres (three and a half inches) from the mid-line. There were no bruits. The systolic blood pressure was 140 and the diastolic pressure 90 millimetres of mercury. The peripheral vessels were soft. No abnormality was found in the lungs or other organs. The pulse rate after exercise was 120 and the respiration rate 32 per minute. The pulse rate returned to 100 in one minute. An electrocardiograph was normal. Fluoroscopic examination revealed a small heart shadow of normal shape, no widening of the aorta, and no abnormality of the lungs. The

transverse diameter of the heart was 12·6 centimetres. The cardio-thoracic ratio was 43%. There had been a previous diagnosis of chronic myocarditis. My diagnosis was *angina innocens* with neuro-circulatory asthenia.

Structural Changes.

This brings us to the second question: If heart disease is present, what are the structural changes? The first important point is to decide if the heart is enlarged, by palpation of the apex beat, or by fluoroscopic screening, if there is doubt.

Next comes the study of the heart sounds. I have already briefly discussed systolic murmurs and mentioned that they are often unrelated to organic disease; but diastolic murmurs, either at the apex or the base, are significant of valvular disease. Let me remind you that in early aortic incompetence the diastolic murmur is very soft and low-pitched, and may be heard on either side of the sternum, often with difficulty and sometimes only if the patient leans forward and stops breathing after a deep expiration.

In the estimation of structural changes consideration is also given to the measurement of the blood pressure and the state of the arteries, including the aortic and coronary vessels.

Functional Changes.

The third question (what are the functional changes?) refers to the various arrhythmias, including auricular fibrillation, flutter and heart block, congestive failure and general vascular (or peripheral) failure. Under this heading I wish first to discuss masked hyperthyreoidism. This, a rather common cause of obscure cardiac symptoms in middle-aged people, is sometimes labelled "chronic myocarditis". The typical picture of Graves's disease seen in younger people is rare. In no less than 10% of proved cases the thyreoid was not palpable (J. H. Towers, *The Lancet*, Volume I, Number 67, 1933). In many patients only a small nodule can be felt. Attacks of palpitation are the main complaint, and during these attacks there is either simple tachycardia, tachycardia with premature beats, or bouts of paroxysmal auricular fibrillation. There is usually a fine tremor; but there are seldom any eye signs, marked vasomotor disturbances, or much loss of weight. Cardiac over-action is the chief sign, and this can be appreciated by palpitation of the apex beat. The first sound is sudden and forceful and may sound blurred through the stethoscope, and this is sometimes mistaken for a presystolic bruit. The over-action causes the apex beat to be diffuse; this may give a wrong impression of enlargement. The general appearance of the patient may be deceptive also. The patient is sometimes apathetic and fat, thus contrasting with the alert, thin, jerky types of younger people with the same disease. In addition the lack of exophthalmos and the absence of much change in the thyreoid gland add to the difficulty. The skin may be warm, however, and moist, and may be pigmented, and nearly all these people have an increased basal metabolic rate. The systolic blood pressure is usually raised, and, if there are no complications, the diastolic pressure is low, giving a characteristic increase in the pulse pressure. The following case illustrates some of these points.

CASE III.—A male, aged fifty-five years, a public servant, complained of epigastric pain and occasional vomiting. The pain was relieved by food and alkalis, and had been present with remissions for three years. The condition was considered to be due to peptic ulcer, and he was kept in bed for three weeks, taking the routine diet and alkalis. At the end of that period he became free of all symptoms and indigestion, but failed to regain his strength. Five weeks after the beginning of treatment he complained of weakness, giddiness and shakiness. No thyreoid enlargement could be detected; but his hands were sticky and tremulous, the skin was warm, and his heart rate 96 per minute after resting. Later the thyreoid was just palpable, and his pulse rate was 104, systolic blood pressure 160 and diastolic pressure 70 millimetres of mercury. There was tremor, his manner was jerky, and the typical salmon colour of the skin was beginning to show on the back of his hands and his face. There were no complaints of palpitation, and the heart was not enlarged, but was found to be over-acting. No eye signs were present, but a slight stare developed subsequently. His basal metabolic rate was found to be +30%. After subtotal thyreoidectomy he made a rapid and complete recovery.

This brings us to auricular fibrillation and its various causes. It is most important to think first of the thyreoid in this connexion, as the condition can be dramatically cured by our surgical colleagues. This illustrates one of the few instances in clinical medicine when an enlarged heart can be observed to revert to its original size. On the other hand, if the condition is unrecognized, congestive failure and death may result.

Another cause of (or perhaps one should say "condition associated with") auricular fibrillation is mitral stenosis. With the advent of fibrillation the presystolic murmur often disappears, and if the heart rate is rapid the diastolic bruit is difficult to hear. A history of rheumatic fever is frequently not obtained. Screening is of great value here; for, if the patient is placed in the right oblique position and examined while he is swallowing some barium, the œsophagus will be seen to be displaced posteriorly by the enlarged left auricle. The œsophagus is also displaced by a widened aorta; but in this case it is carried to the left as well as posteriorly, as may be seen in antero-posterior views (*American Heart Journal*, June, 1937).

A third cause of auricular fibrillation is hypertensive cardiac failure. If the patient is first seen after congestive failure has occurred, the ætiology may not be at once evident, for the blood pressure often, but not always, falls. The relatively high diastolic pressure may give the clue, together with enlargement of the heart, chiefly involving the left ventricle, perhaps with a systolic bruit at the apex, signifying relative mitral incompetence. The peripheral arteries may be thickened and changes may be seen in the retinal vessels. The past history often helps.

Fourthly, auricular fibrillation may occur paroxysmally in the absence of demonstrable organic cardiac changes. This is less common than the

types already mentioned, and is regarded as a functional disorder. It is amenable to treatment with quinidine.

Other very rare causes include pneumonia, mental disease, cholecystitis and dental sepsis.

Ætiology.

On the importance of the fourth question (what is the ætiology?) hangs the whole of my argument. For clinical purposes chronic myocarditis, unqualified, does not exist. It is analogous to a diagnosis of colitis when used to cloak one's ignorance of the cause of some abdominal discomfort. In every case of heart disease an attempt to find the cause should be made, and in nearly every case it can be discovered, or at any rate inferred.

In Table I is a list that gives some idea of the relative frequency of the various factors in my own private practice. It has been compiled from a study of 435 patients suffering from symptoms or signs referable to the cardio-vascular system. They are given in the order of frequency.

Table I.

The Causation of Symptoms Referable to the Cardio-Vascular System in a Study of 435 Patients.

Diagnosis.	Number of Cases.	Percentage.
Hypertension without demonstrable cardiac disease	161 } 240	37·0 } 56
Hypertensive heart disease	80	19·0
Functional heart disease	88	20·0
Coronary disease (including *angina pectoris*, coronary occlusion and bundle branch block)	32	7·3
Thyreotoxicosis	29	6·6
Rheumatic heart disease	18	4·0
Syphilis (aortitis and aneurysm)	7	1·6
Cor pumonale	6	1·3
Nephritis	5	1·2
Congenital *morbus cordis*	4	0·9
Unclassified	3	0·7
Subacute bacterial endocarditis	1	0·2
Arterio-venous aneurysm (with enlarged heart)	1	0·2
Total	435	100

You will notice the large preponderance of patients with hypertension, and when dealing with congestive failure it is this group again, as one would expect, that heads the list. The functional group comes next in frequency, and I want particularly to draw your attention to this fact. You are already aware of the prevalence of thyreotoxicosis in this town. The rheumatic group is small and, I feel sure, is not a true indication, and an analysis of the hospital figures, which it is hoped to produce later, is expected to reach a higher percentage. Coronary disease should probably show a higher figure.

With regard to cardio-vascular syphilis, I would remind you of the difficulty of detecting aortitis and aneurysm in the early stages, unless the possibility is kept in mind, and of the value of fluoroscopic screening in its detection. Only about one-third of these cases is complicated by aortic insufficiency; but when the latter is found in a person of middle age, a Wassermann test should be carried out, even if hypertension and arteriosclerosis are present, as they are frequently present together with cardio-vascular *lues*.

The incidence of nephritis is known to be low in Tasmania. Please note the absence from the list of chronic myocarditis, fatty heart and primary heart strain. Most cardiologists have abandoned these three conditions as clinical entities.

With regard to fatty heart, we know that obesity interferes with the heart's reserve by mechanical means, chiefly by raising the diaphragm, as already described. There is no evidence that fatty infiltration of the heart, *per se*, actually has any effect on the myocardium. Physical strain in a person with a previously healthy heart does not cause organic damage. Such experienced men as Mackenzie and Lewis have stated that they have never seen a normal heart structurally damaged by exertion. Recently, extensive investigations have been made on marathon runners and oarsmen, and the results all tend to support this view. My own cases have proved to be due to anxiety, which, acting through the autonomic system in susceptible people, causes neuro-circulatory asthenia. Here is a typical example.

Case IV.—A labourer, aged twenty years, had never been ill before. When attempting to force back into place a truck of concrete, which had become unhooked, he experienced a severe pain in the left side of the chest. He thought he must have strained his heart, and this fear was confirmed by the doctor who examined him. After several weeks' rest he attempted to start work; but the pain returned and he became short of breath on the least exertion and had to give up. He made a third attempt at work with the same result. I first saw him ten months after the accident, when he complained of a typical "effort syndrome": shortness of breath on exertion and palpitation and chest pains. He felt nervous and exhausted. On examination the heart rate was found to vary from 80 to 100 per minute, and premature beats occurred occasionally. His heart was not enlarged, the apex beat being in the fifth intercostal space, 8·1 centimetres (three and a quarter inches) from the mid-line. The sounds were forcible. The systolic blood pressure was 135 and the diastolic pressure 85 millimetres of mercury. Electrocardiography showed an occasional ventricular type of premature beat, with low voltage and split *Q R S* complex in Lead III and a negative (inverted) *T* wave in Lead III. Screening revealed a normally shaped over-acting heart and normal lungs. His basal metabolic rate was +8%. After firm reassurance and persuasion and graduated exercise he made good progress, so that in a fortnight he could walk six miles without symptoms. He has now been back at work for ten months without relapse.

Let us now return to the question of diagnosis in a patient who has complained of symptoms suggestive of heart disease. Having answered the four questions, and bearing in mind the above remarks, one should be in a position to make a complete diagnosis. If there is still the least doubt, the heart should be examined by the fluoroscopic screen and an electrocardiograph should be taken; for the patient must not be left with a casual remark about myocarditis or a "flabby" heart.

Anyone who has attempted the study of heart disease must be struck by the immense importance of the psychological effect on the patient of what he

is told about his heart. I shall never forget how incensed I was with one of my colleagues.

I had spent a long time in making a careful examination of a patient, and a longer time in persuading him that his heart was not diseased, and he went home well on the way to recovery. The next time he called I was out and he sought advice elsewhere and was told he had myocarditis. When I saw him again he was far worse than at the first visit and was certain the end was not far off. It has taken me nearly a year to obliterate the effect of those casual words.

The case of returned soldiers receiving pensions for myocarditis, whether they have diseased hearts or not (and often they have not), seems almost hopeless. If a man has been convinced that he has had a bad heart for twenty years, it must require the full resources of a competent psychologist to persuade him out of it; but it is within the power of all of us to prevent this disaster from occurring in civil life. If a small doubt remains in the physician's mind as to organic disease, surely he should have the courage to take the risk to his reputation and spare the patient, who probably associates any type of heart disease with the possibility of sudden death. However, one must not err in the opposite direction and diagnose functional disease too readily. Let me relate another case to act as a curb to over-enthusiasm in this respect.

CASE V.—A female, aged fifty-seven years, complained of pains in her chest, mostly in the left submammary region. The symptoms were of six weeks' duration. The pains were present at rest, but were aggravated by exertion, which also caused some dyspnœa. These pains were often sharp and caught her breath, and she was subject to palpitation and was easily exhausted. Her previous history was uneventful.

She looked well and there was no dyspnœa. Her colour and nutrition were both good. The heart rate was 96 per minute and there were occasional premature beats. So far the picture was typical of *angina innocens*. The apex beat, however, was a good 10·0 centimetres (four inches) from the mid-line, in the fifth intercostal space, and there was a soft systolic murmur at the apex. On careful listening while she leant forward after a deep expiration a very soft, low-pitched diastolic blow could be heard at the left border of the sternum opposite the third interspace. The pulses were equal, with a tendency to the Corrigan type, which was confirmed by finding a pulse pressure of 110 millimetres of mercury. The systolic blood pressure was 170 and the diastolic pressure 60 millimetres of mercury. The precordial muscles were tender to pressure. An electrocardiograph showed occasional auricular premature beats and negative *T* wave in Lead III; but screening revealed a widened aorta in all three views and an enlarged heart shadow. The transverse diameter measured 14·2 centimetres and the cardio-thoracic ratio was 60%. The patient reacted (+++) to the Wassermann test. The diagnosis was luetic aortitis.

In cases of organic disease the psychological aspect is equally important, and unnecessary restrictions should not be imposed. My own view is that the full prognosis should only in very exceptional circumstances be divulged to the patient, the relatives, of course, being taken fully into one's confidence.

Summary.

One might summarize the whole of the foregoing remarks in a few words. A patient who complains of some shortness of breath, vague chest pains and palpitation or irregularity of his heart beat, and perhaps has the added misfortune of a systolic murmur at his apex, has a 20% chance that no organic disease is present, and a 100% chance that he is not suffering from chronic myocarditis.

Reviews.

OBSTETRICS AND GYNÆCOLOGY.

THE seventh edition of Bourne's "Synopsis of Obstetrics and Gynæcology"[1] is an excellent book of its kind. The mere fact that a seventh edition has been required is a sure indication that the book is serving its purpose and is providing the student and practitioner with a means of revision of the subjects covered. These include normal and abnormal pregnancy, normal and abnormal labour, the puerperium and the new-born child, and obstetric surgery; while in Part II of the volume there is a fairly complete synopsis of gynæcology covering most of the ills found in the female pelvis. There is, of course, no attempt at any literary style; the essentials of physiology, pathology, symptoms and physical signs are set forth in tabular form for each condition. The sections on treatment are very well handled, being at the same time concise, adequate and up to date. The uses of mandelic acid and "Prontosil" are described in their appropriate places, and the physiology of the sex hormones is briefly but intelligibly outlined. The senior student wishing to revise the subjects covered in this volume, will find it invaluable for the purpose. The addition of a bibliography would make it of more value to the practitioner.

THE PLANNING OF EXPERIMENTS.

THE welcome given to the first edition of Dr. Fisher's volume on the application of statistical methods to the planning and interpretation of experiments in the fields of medicine, biology and related subjects left no doubt that a second edition would shortly be called for. The new edition of "The Design of Experiments" is now available.[2] Apart from numerical corrections the principal changes have been made in the section dealing with completely orthogonalized squares, which are now treated more fully. Some examples have been added to the chapter on "Confounding". In many fields of experimentation there is a lack of quantitative knowledge about the degree of heterogeneity to be anticipated in batches of material of different size, or drawn from more or less diverse sources. The means by which such heterogeneity can be mathematically controlled are described and illustrated.

At the present time, when large scale experiments on animals are engaging the attention of many research workers, the chapter on the increase of precision by concomitant measurements is to be specially commended. For example, in a feeding experiment with animals, when their response to a number of different diets is to be evaluated, it is often possible to ensure that the animals undergoing the test shall be of the same age and often also that they shall be closely related or of the same parentage. But it may be that the differences in initial weight constitute an uncontrolled cause of variation among the responses to

[1] "Synopsis of Obstetrics and Gynæcology", by A. W. Bourne, M.A., M.B., B.Ch., F.R.C.S., F.C.O.G.; Seventh Edition, fully revised. Bristol: John Wright and Sons Limited. Crown 8vo, pp. 456, with illustrations. Price: 15s. net.

[2] "The Design of Experiments", by R. A. Fisher, Sc.D., F.R.S.; Second Edition; 1937. Edinburgh and London: Oliver and Boyd. Demy 8vo, pp. 272. Price: 12s. 6d. net.

treatment; this will detract from the precision of the comparisons. The author describes what bearing a random choice of the animals has on the tests of significance and how the factor of weight may be eliminated. The effects of the initial weights upon the final results may approximately be estimated and the results adjusted in accordance with the estimated effects, so that the final precision in many cases is almost as great as though complete equalization had been possible.

Biological experimenters will do well to peruse carefully the author's remarks in the opening section of the chapter on the "Factorial Design in Experimentation". It is pointed out that in expositions of the scientific use of experimentation it is frequently found that an excessive stress is laid on the importance of varying the essential conditions only one at a time. This may be justifiable in experiments merely designed to demonstrate simple laws, connecting cause and effect; but in the state of knowledge or ignorance in which genuine research, intended to advance knowledge, has to be carried on, this simple formula is not very helpful. We are usually ignorant as to which out of innumerable possible factors may prove to be the most important. We have usually no knowledge that any one factor will exert its effects independently of all others that can be varied, or that its effects are simply related to variations in those other factors.

Dr. Fisher's work seems destined long to remain the standard work on the planning of large-scale experiments.

MEDICAL STATISTICS.

THE editor of *The Lancet* writes in his foreword to Dr. Hill's book on medical statistics: ". . . in clinical medicine today there is a growing demand for adequate proof of the efficacy of this or that form of treatment. Often proof can come only by means of a collection of records of clinical trials devised on such a scale and in such a form that statistically reliable conclusions can be drawn from them. However great may be our aversion to figures we cannot escape the conclusion that the solution of most of the problems of clinical or preventive medicine must ultimately depend on them."

Acting on the belief that there is now a steadily increasing demand among both clinical and public health workers for some knowledge of statistical technique, the same editor invited Dr. A. Bradford Hill to prepare for *The Lancet* a series of short simple articles on such methods as his experience of medical statistics had shown would be most useful in that field. Dr. Hill has succeeded admirably in making clear, even to the non-mathematically minded, the kinds of error they are most likely to make and how they may be avoided. At the request of many readers of *The Lancet* these articles have now been reissued in book form under the title "Principles of Medical Statistics".[1]

The statistical method is required to interpret figures which are at the mercy of numerous influences; one of its objects is to determine whether individual influences can be isolated and their effects estimated numerically. The essence of the method consists in the comparison of like with like and in taking into consideration any relevant factor in, say, two groups of experimental cases.

The author takes the reader by a simple route through the ordinary routine work, such as the presentation of statistics, the variability of observations, calculation of the standard deviation, problems of sampling, coefficient of correlation and life-tables and survival after treatment. Three chapters at the end of this valuable little book deal with common fallacies and difficulties. Some of these fallacies are amusingly treated. The author states that Farr pointed out three-quarters of a century ago that the average age at death of bishops is much higher than the corresponding average of curates. But making all the curates bishops will not necessarily save them from an early death. The average age at death in an occupation depends, in part, on the age of entry to that occupation and the age of exit from it, if exit takes place for other reasons than death. Bishops have a higher age at death than curates because few men become bishops before they have passed middle life, while curates may die at any age from their twenties upwards.

In contrast with the more advanced book of Dr. Fisher this little volume is easy to read and highly informative. The reader will find the illustrative examples both striking and amusing. Each chapter is followed by a summary which recapitulates the main points of the argument. The last chapter gives a general summary and outlines the conclusions that have been arrived at. The final section bears the appropriate title "Commonsense and Figures".

The Lancet, no less than the author, is to be congratulated on making this simple course on statistics available not only to the medical practitioner, but also to the general public.

[1] "Principles of Medical Statistics", by A. B. Hill, D.Sc., Ph.D.; 1937. London: The Lancet Limited. Demy 8vo, pp. 179. Price: 6s. net.

MATERIA MEDICA AND THERAPEUTICS.

HALE-WHITE'S "Materia Medica, Pharmacology and Therapeutics" is so well known and appreciated that it has now attained its twenty-third edition.[1] This edition includes all the essential alterations embodied in the 1936 Addendum to the British Pharmacopœia of 1932. Special note is directed to the changes in the official doses of cinchophen (the maximum dose being reduced to 10 grains), *Ferri et Ammonii Citras* (the maximum being increased to 40 grains) and cod liver oil, which is diminished. Additions include benzedrine. It is stated that this drug is used for narcolepsy, depressive psychoses, arterial hypotension and spasmodic conditions of the digestive tract. Contraindications are hypertension and other cardio-vascular disease. It is mentioned that precordial pain and even collapse have been induced by it in seemingly healthy subjects. Digoxin from *Digitalis lanata* receives brief but explicit notice. The chapter on ergot has been rewritten. The composition of ergot is now intelligibly discussed, due consideration being given to ergometrine. The bewildering behaviour of the liquid extract of ergot of the 1914 and the 1932 editions of the Pharmacopœia has been explained by the discovery of the alkaloid, ergometrine. The recently isolated alkaloids sensibamine and ergoclavine are also referred to. Regarding the use of ergotamine for migraine, it is mentioned that central vomiting, rise of blood pressure and bradycardia may follow the injections. Incidentally the use of ergot for internal hæmorrhage is rightly stated to be futile or dangerous. Histamine is fully dealt with. As regards histidine for peptic ulcer, it is pointed out that evidence of its efficacy is conflicting and that its mode of action is not understood. When benefit has occurred, relapse soon follows discontinuance of the drug. Concerning the use of mandelic acid in urinary infections, it is held that the results in uncomplicated cases are excellent, but that it must not be given in severe renal disease with raised blood urea. Other matters mentioned are protamine insulate; the work of Hoyle and Evans on nitrites; the use of physostigmine and prostigmin in *myasthenia gravis*; the observations of Butsch, McGowan and Walters regarding the action of morphine on the biliary system, and a most valuable paragraph on bromide intoxication. Altogether the work is thoroughly up to date and reliable. We miss any reference to sulphanilamide ("Prontosil"), and a few misprints are noticed; but the general excellence of the work is well maintained, and it continues to be a valuable text-book for students and an excellent work of reference for practitioners.

[1] "Materia Medica, Pharmacy, Pharmacology and Therapeutics", by W. Hale-White, K.B.E., M.D., LL.D., revised by A. H. Douthwaite, M.D., F.R.C.P.; Twenty-Third Edition; 1937. London: J and A. Churchill Limited. Crown 8vo, pp. 574. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 6, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

TUBERCULOSIS.

The last three issues of this journal have been devoted to an account of the proceedings of the fifth session of the Australasian Medical Congress (British Medical Association). Everyone who attended voted the gathering successful from every point of view. The choice of tuberculosis as the motif of the congress was justified by the many aspects of the subject brought before the several sections and by the importance of the information placed before the plenary session on tuberculosis. The members of the medical profession in Australia have every reason to be grateful to the three opening contributors to the discussion, and especially to Dr. M. J. Holmes for the enormous amount of work that the preparation of his paper entailed. If the lead that was given at the first plenary session is followed, we make bold to state that this discussion alone will have made the holding of the fifth session worth while.

The investigations made by Dr. Holmes show that the mortality rate in Australia, which reached its highest point at about the year 1885, has been falling without interruption since that time. The rising rate prior to 1885 was associated with an acute form of the disease, causing high mortality in childhood and early adult life, at a time when the population contained an abnormally large proportion of young persons. This mortality rate, as Dr. Holmes pointed out, has given place to a rate which reaches its peak as far as males are concerned in the age group fifty-five to sixty years; the curve for the mortality rate for females almost reaches its peak in the age group twenty to twenty-five years. In criticizing his own paper, Dr. Cotter Harvey expressed the opinion that the high incidence in young females was due *inter alia* to child-bearing, industrialization and the stress of modern life. It would be quite simple to enlarge upon this statement. How far child-bearing is a factor may be questioned in these days of the falling birth rate, but without doubt industrialization is largely responsible, particularly because young women employed in industrial undertakings insist on taking their part in modern social life. They work hard and give too much time to social scamperings, spending too much on their clothes and too little on their food; possibly it would be better to state that what they spend on their food is not spent wisely. This may not, however, be the whole story, because in South Australia the mortality from tuberculosis among young women is much greater than in any of the other Australian States; in the twenty- to thirty-year age group it is double that of New South Wales in the same age group. There is no reason to suppose that industrial and social conditions are more exacting in South Australia than in any of the other States. In other words, Australia is confronted with the tuberculosis problem common to other countries, but it has a peculiar complication of that problem. Obviously, if the problem peculiar to South Australia could be solved, light might be thrown on the general tuberculosis problem.

In the Adelaide discussion stress was laid on the pre-clinical or subclinical manifestations of the disease and it was made clear that more attention should be paid to this aspect of the subject. Dr.

Holmes stated that more consideration should be given to prevention than to other aspects of the problem. At the same time he stated that already millions of pounds had been and were being saved by the progress in the control of tuberculosis. Of the utmost importance is the recommendation that provision should be made by a system of allowances for the nutrition of families of patients suffering from tuberculosis. There is also no doubt that in each State there should be, as Dr. Holmes holds, a governmental officer in charge of a division of tuberculosis, who should devote his whole time to directing and coordinating the work of clinicians in the fight against the ravages of the tubercle bacillus. If such an officer begins to dabble in treatment and in the care of individual patients he will lose his perspective, and unless he is a man of extraordinary ability and is possessed of unusual power of detachment, he will eventually become ineffective. From what we can gather the congress discussion is likely to have immediate and far-reaching results. The National Health and Medical Research Council will meet before long and the question of tuberculosis and its control will probably be considered. It is likely that an effort will be made to determine whether the infection rate in tuberculosis is diminishing by the carrying out of Mantoux tests in groups of children and adolescents; also a survey will be made in certain industrial groups to ascertain the reason for the high mortality among young women. It is to be hoped further that work will be directed towards determining the strain of tubercle bacillus, whether human or bovine, associated with various non-respiratory tuberculous lesions in human beings. The council has at its disposal funds that have been provided by Parliament; no more useful field for their expenditure could be found.

Current Comment.

TRANSFUSION OF STORED CADAVER BLOOD.

The conception, economical if gruesome, that the blood of a corpse should be collected, stored and reinfused into living man is one which may, with some confidence, be divined as originating in the East. It happens to have received most attention at the hands of Professor S. S. Yudin, chief of the surgical clinic of the Post-Graduate School at Moscow.[1] He had unique opportunities for pursuing his ideas, as he had complete charge of an emergency hospital admitting from eight to ten thousand patients every year. Each day would provide sufficient dead or moribund persons to ensure a supply of fresh cadaveric serum and corpuscles for transference to a more needy subject. Experimental work on dogs had previously allayed the fear of "toxic" reactions, which it had been supposed would follow the reemployment of such blood. The Wassermann reaction, however, had to be carried out in the shortest possible time. The preservation of the complementary powers of serum after death is well known and is frequently made use of in routine autopsy investigation. Professor S. S. Yudin's laboratory colleagues were successful in shortening the time factor necessary for a reliable interpretation of the reaction to a period of four hours. Even this delay, however, was, under conditions of urgency, far too great, and transfusions often had to be undertaken with all dispatch and at the risk of transference of syphilis. The legal and ethical considerations of commencing such a procedure were on the one hand the risk of death from the accident, and on the other the question of the formerly feared "cadaveric intoxication", a possible syphilitic infection, and the certainty that even if the patient died from the shock of the accident the blame would be attached to the transfusion. Yudin's account of his first attempt is worthy of a Guignol setting. The victim was a healthy young suicide on the point of death from severance of his radial arteries. The donor was an elderly gentleman already dead for twenty-four hours owing to a fractured skull. About 420 cubic centimetres of cadaver blood were obtained from the inferior *vena cava*, after massage and posturing of the limbs, and this, mixed with about half its volume of normal saline solution, was infused into the young man with life-saving effect. Further experimental work on dogs proved that the foreign hæmoglobin enhanced the oxygen carrying power of the recipient's blood. Apparently the erythrocyte of fresh cadaver blood completely regains its rôle as an oxygen carrier. Subsequently blood was collected from the jugular vein, with the cadaver in the Trendelenburg position. The yield from a cadaver from which little blood has been previously lost varies from 900 to 1,500 cubic centimetres. It was found that refrigeration of stored blood preserved the properties of such blood intact, besides providing sufficient time for serological and bacteriological safeguards to be undertaken. The use of citrate is unnecessary, as after sudden death the blood remains liquid after autofibrinolysis. A temporary clot forms, but redissolves after an hour or so. In persons dying from severe or chronic

[1] *The Lancet*, August 14, 1937.

disease the blood behaves like that of living people. The use of fibrinolysed blood offers a further guarantee against the risk of transmission of syphilis or other chronic infections, and allows of the transfusion of pure whole blood without any diluent.

Over one thousand of these transfusions have been effected by Professor Yudin and his staff. Errors of technique have resulted in five fatal complications in the series. Twice deep jaundice developed, but the patients survived. The type of patient selected was either a sufferer from cancer upon whom some radical surgical procedure was contemplated or performed. Acute internal hæmorrhages and grave traumatic shock found further large groups. The adoption of the large drip transfusion of Marriott and Kekwick is stated to have improved further the results following profuse gastric hæmorrhage.

The adventurous spirit of this work has had its reward in the saving of many Russian lives. The advantages of immediately using unlimited quantities of stored whole blood in either civil or military emergencies need no elaboration. The distasteful and dangerous aspects of the procedure can apparently be overcome. The presence of a central emergency hospital, to which all serious accident patients were sent, would be an indispensable factor in the accumulation of supplies of blood. When the technical difficulties of collection and the small but inescapable risks of reaction and infection are further added, the method would be unlikely to supplant an adequately organized twenty-four-hour blood transfusion service, such as is available in London and is long overdue in certain large Australian cities.

GANGRENE CAUSED BY ERGOTAMINE.

S. PERLOW AND L. BLOCH remind us that ergotism is manifested in two ways: the gangrenous and the convulsive types.[1] The gangrenous form usually affects the extremities, and fingers and toes may be lost or even an entire limb. The convulsive type is considered to be due to an action on the central nervous system, and its manifestations consist of depression, weakness, headache and eventually convulsions, both tonic and clonic. Epidemics of chronic ergotism have been described in eastern Europe for centuries following the consumption of rye bread infested by ergot. In the United States of America the last epidemic of this kind occurred in New York in 1825. Acute ergotism is more rapid in onset and development, but otherwise resembles the chronic form. It has occasionally followed the therapeutic employment of ergot or its derivatives, particularly ergotamine tartrate. The smallest recorded quantities of this substance producing gangrene have been one milligramme given subcutaneously over a period of four days and twenty-six milligrammes taken by mouth in a week. The treatment of gangrenous ergotism has been unsatisfactory and no instance of complete cure by any method of treatment is on record. Benefit has been reported from theophylline and also from hyoscine. Adrenaline has been stated to abolish the ergotamine effect and has been used, but without benefit. This is only to be expected from a knowledge of the pharmacology of each substance. M. E. O'Sullivan last year reported a large series of cases in which ergotamine was employed for migraine and untoward effects were considered to have been averted by the use of atropine and calcium.

Perlow and Bloch report a case of ergotamine tartrate poisoning, with impending gangrene of both feet. In this case rapid and complete cure, without actual gangrene, followed the use of papaverine hydrochloride. The patient suffered from Hodgkin's disease with jaundice and generalized pruritus. For this pruritus ergotamine was administered. In all, the patient received 1·0 milligramme of ergotamine tartrate by mouth and 2·5 milligrammes hypodermically. Intense pain in the toes ensued. The toes became cold, slightly œdematous and cyanotic. It was considered that there was spasm with possibly commencing organic occlusion of the arteries of both feet, with impending gangrene. Treatment with papaverine hydrochloride was instituted with complete recovery and without any remaining discoloration, impairment of sensation or loss of tissue. Perlow and Bloch observe that in the gangrene of ergotism there is a primary vascular spasm followed by thrombosis due to injury of the intima and stasis. They had previously obtained good results from the use of papaverine hydrochloride as an antispasmodic in other forms of peripheral circulatory disturbance; hence their employment of the drug in this instance. W. M. Yater, however, had already recommended this drug for bilateral gangrene of the feet due to the use of ergotamine tartrate for the pruritus of jaundice. Perlow and Bloch consider that in this case the results were so dramatic as to leave no doubt in their minds as to the efficacy of the drug as a vascular antispasmodic. The value of papaverine hydrochloride as an antispasmodic in other types of peripheral arterial disease was already well known, and the authors consider their case as furnishing additional evidence of the value of this drug in relaxing arteries in a condition of spasm. It may be stated that papaverine is only a comparatively weak poison, without the soporific effect of morphine. It apparently acts directly on the blood vessels, causing dilatation, without materially affecting the blood pressure. It apparently does not engender addiction. In certain coastal areas of New South Wales a form of ergot attacks paspalum grass. It would be of great interest and value to have the pharamcology of this species completely elucidated to ascertain how it compares with the pharmacopœial species.

[1] *The Journal of the American Medical Association*, July 3, 1937.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

The Friedman Test in the Pregnant Rhesus Monkey.

THE Friedman pregnancy test, which has proven so reliable for the human being, has almost uniformly failed when tried on other animals, the chimpanzee only having given positive reactions. The macaque has heretofore yielded only negative results, Snyder and Wislocki having tested the urine from this monkey from the end of the first month of pregnancy to parturition without ever securing a positive result to the test. Because of an idea that in the monkey the excretion of prolan in the urine might be a transitory process, occurring only during the establishment of the placenta, G. W. D. Hamlett (*American Journal of Physiology*, April, 1937) made a series of tests on pregnant macaques in the Carnegie colony at Baltimore, starting the testing as early as possible during the animal's pregnancy. These tests show that the pregnant monkey gives a perfectly typical positive Friedman reaction for a period of about a week soon after implantation. The author considers that the failure of other investigators to induce ovulation in rabbits by the injection of monkey pregnancy urine is undoubtedly due to their having started the tests too late to detect the transitory presence of the urinary prolan. Positive reactions were secured only during the nineteenth to twenty-fifth days following ovulation and conception. In several series of tests the monkeys eventually proved to be non-pregnant; these tests uniformly gave negative results. Three out of five pregnant monkeys gave positive results to the test; of the remaining two, one tested only at the eighteenth day gave a negative result; the other negative result was obtained from a monkey which was not tested until the twenty-fourth day. This extremely transitory excretion of the urinary prolan compares quite closely with the curve of its output in the pregnant woman. In the woman, however, while there is a tremendous drop in the number of units of prolan excreted, and while this drop occurs at a time (about the eighth postmenstrual week of gestation) somewhat comparable with that in the monkey, the base level of prolan output remains high enough throughout the remainder of pregnancy, so that five cubic centimetres of unconcentrated urine will always yield a positive result in the rabbit. In the macaque, on the other hand, the output drops to such a low level that twenty cubic centimetres of urine, after the twenty-fifth day, do not contain even a single rabbit unit of prolan. The situation in the apes is not yet clear. Schultz and Snyder obtained positive reactions from the urine of pregnant chimpanzees at 95 and 100 days, post-menstrual computation, and negative results from day 160 onwards. It appears, on the basis of present knowledge, as if we might be dealing here with a phylogenetic series in the evolution of a physiological character. The excretion of detectable quantities of prolan, which is such a transitory phenomenon in the catarrhine monkey, becomes a three-months-old process in the ape, while in the human it has developed into a physiological characteristic of such stability that it persists throughout gestation from its first appearance, shortly after implantation, and disappears only after parturition.

Calcium Concentration and the Coagulation Time of Blood.

WHILE calcium is recognized as essential for normal coagulation, there is no evidence in the literature of the exact amounts of calcium required. Such studies as have been made deal for the most part with blood treated with oxalate or other anticoagulants. Since the mechanism by which these anticoagulants, particularly oxalate, produce their effect is not entirely clear, any calculations as to the quantity of available calcium in blood so treated must be somewhat uncertain. Clinical and animal investigations for the most part have offered no grounds for believing low blood calcium to be a cause of increased coagulation time. Marian M. Crane and Heyworth N. Sanford (*American Journal of Physiology*, April, 1937) present a series of experiments in which the influence of variations in total calcium concentration upon the coagulation time of blood was determined. To avoid confusion resulting from the addition of anti-coagulants, these experimenters made use of untreated whole blood. Since the rôle of calcium in coagulation is in the conversion of prothrombin into thrombin, it is important that no platelet disintegration occur to initiate thrombin formation before the desired experimental conditions are produced. The blood was therefore drawn from normal adults into chilled oiled syringes and transferred immediately to paraffined tubes standing in ice. It was determined in preliminary experiments that in a dilution of 1 in 10 with normal saline solution blood would not coagulate; but if calcium was added, a firm clot was formed. This proportion of blood and diluting solution was consequently adopted for the experiments. It was found that coagulation time was practically constant with calcium concentrations between 5 and 20 milligrammes *per centum*. Marked prolongation of coagulation time appeared first with calcium reduced to 2·5 milligrammes *per centum* or less. A second series of experiments showed that variations in amount of plasma protein present, which might alter the proportions of diffusible and non-diffusible calcium, did not affect the relationship between total calcium and coagulation time. In view of the results, these authors consider that prolonged coagulation time as observed in hæmorrhagic conditions cannot be interpreted as ever being the result of low blood calcium alone.

The Lactogenic Hormone of the Anterior Pituitary Gland.

WORK by Grüter and Stricker, Corner and Riddle and Bates has demonstrated a lactogenic principle in the anterior pituitary gland. Riddle and Bates have presented data indicating that the lactogenic substance may be a separate and distinct principle of the anterior lobe. Everett Idris Evans (Proceedings of the American Physiological Society, *American Journal of Physiology*, June, 1937) describes the preparation of the lactogenic principle by a new method of extraction, precipitation and separation from the gonadotropic and thyreotropic factors. Preparations have been made that assay as high as 33 units per milligramme. The new lactogenic preparation of the anterior pituitary will stimulate milk secretion in rabbits at one milligramme per kilogram. By the use of from 1,000 to 2,250 units lactation has been induced in six nulliparous and multiparous human females, and gynæcomastia has been produced in one male. The author reports that the lactogenic principle is apparently a protein substance, reacting strongly to the biuret, anhydrin, xanthoprotein and Hopkins-Cole tests, and failing to react to Koessler-Hanke and Millon reagents. There are indications that the present preparations are rich in tryptophane, but contain no tyrosine. There is a highly labile sulphur group in the impure preparations, but none in the purer preparations. There is about 3·5% cystine in those preparations from which 25 units per milligramme are assayed. In all but one experiment this new lactogenic preparation was destroyed by boiling for one hour at pH 7·8 to 8·2. This particular lactogenic preparation contains no vaso-dilator substances.

A Study of After-Contraction.

THE phenomenon termed after-contraction is shown in the following manner. When any set of muscles, such as those involved in raising the extended arm or leg, is voluntarily kept in action for a period of time and then voluntarily relaxed, there occurs involuntarily a repetition of the original movement, accompanied by a sensation of lightness or floating upward of the part. This phenomenon has long been known, and various studies of it have been made in animals which have been decorticated or decerebrated, or were under the influence of anæsthetics. M. R. Sapirstein, R. C. Herman and G. B. Wallace (*American Journal of Physiology*, July, 1937) have studied this phenomenon in normal human beings. The experimental procedure employed was flexion at the hip. The subject

stood on a platform, supporting his weight on one leg, the other hanging free, with balance maintained by the arm on the supporting leg side resting lightly on the arm of a chair. To an ordinary riding spur fastened to the heel of the free hanging leg was attached a cord passing over two pulleys and ending at a lever, arranged to record the leg movement on a kymograph. Also attached to the spur was a second cord passing over a pulley and connected at its other end with a receptacle in which various weights could be placed. This cord could be disconnected without delay. In these experiments the mechanism which gave rise to the after-contraction was much more complicated than in animal studies on reflex activity or cortical stimulation. When the extremity was raised and immediately relaxed, no after-contraction occurred. If the contraction period was prolonged, the after-contraction appeared, its extent increasing with increase in the duration of elevation. When the strength of stimulus (that is, the weight lifted) was increased, the after-contraction also increased, but not linearly, as saturation was soon reached. When the extremity, with no load attached, was raised and immediately relaxed, no after-contraction occurred. If the cycle was repeated with no delay, the after-contraction might appear after as few as three elevations. Various muscular contractions had a variable reinforcing effect on the after-contraction. The phenomenon was also affected by voluntary inhibition, by fatigue, by a latent period, and variations in the individual subject. In contrast to the tonic contraction, there may be the "epileptoid" or clonic after-contraction in which the rise is not steady but jerky. These authors consider that the characteristics of after-contraction, as described by them, correspond to that of after-discharge, which is a property of neurones, motor or sensory, and which manifests itself in continued activity after cessation of the original stimulus. Muscle itself does not possess this property. It has been described as an enduring central excitatory state. There seemed no doubt that in after-contraction, as studied here, the central link in the chain of events was a persistent excitatory state in the cortical motor neurones. The involuntary movement was in fact a replica of the coordinated volitional movement necessary for its production. These authors suggest that after-contraction is a cortical function entering into many of the phases of coordinated muscular movements.

BIOLOGICAL CHEMISTRY.

Hæmoglobin Regeneration in Nutritional Anæmia.

P. B. PEARSON, C. A. ELVEHJEM AND E. B. HART (*Journal of Biological Chemistry*, July, 1937) have studied the effect of nine different proteins on the rate of hæmoglobin regeneration in nutritional anæmia of the rat. When the intake of iron and copper was adequate to permit hæmoglobin formation at the maximum rate, no special hæmatopoietic property, in addition to their protein content, could be ascribed to liver, casein, egg albumen or soya bean oil meal. All these were consistently effective in building hæmoglobin. No evidence was obtained to indicate that liver was more effective for hæmoglobin formation than were other proteins of good quality. Corn gluten meal and wheat gluten were poorly utilized for both hæmoglobin formation and growth. Animals fed on gliadin and gelatin, which were inadequate for growth, did not regenerate hæmoglobin as rapidly as those fed on proteins which permitted growth. It was shown that, provided adequate iron and copper were given, the animal utilized its own body proteins for hæmoglobin formation when the diet was deficient.

E. B. HART, C. A. ELVEHJEM AND G. O. KOHLER (*Journal of Experimental Medicine*, August, 1937) have presented data which showed that the effectiveness of whole liver in the treatment of nutritional anæmia induced in rats by a milk diet was proportional to its available iron and copper content. Commercial preparations of liver products varied greatly in their hæmoglobin-regenerating efficiency in the anæmic rat, and this variation was correlated directly with the iron and copper content of the preparation.

Carbohydrate and Ketone Utilization.

I. ARTHUR MIRSKY AND R. H. BROH-KAHN (*American Journal of Physiology*, August, 1937), working with nephrectomized and completely eviscerated rabbits, find that the administration of carbohydrate does not influence the utilization of ketone bodies and that the presence of a ketonæmia does not impair the utilization of carbohydrate. They consider that the administration of glucose does not increase the utilization of ketones either by the liver or the extra-hepatic tissues (that is, it is not ketolytic in action) and that the effect of glucose in inhibiting diabetic and non-diabetic ketosis must be due to an actual inhibition of ketone formation (that is, a true anti-ketogenic effect). It is postulated that ketone bodies are end-products of fat metabolism in the liver.

Hæmolysis of Red Cells.

ERIC PONDER (*Journal of Experimental Biology*, July, 1937) discusses the area and volume changes of mammalian red cells under various conditions. Red cells placed in hypotonic media swell to a critical volume, after which hæmolysis occurs. Shortly before reaching this critical volume, the cell becomes a perfect sphere and in hypotonic serum the area of the sphere is substantially the same as the area of the original diskoid red cell in an isotonic medium. Thus the increase in cell volume is not accompanied by an increase in cell surface, but rather by a change in cell shape, and lysis occurs when the volume has increased to such an extent that the cell membrane is subject to stretching forces. The volume increase which a cell can undergo by becoming a sphere with the same surface area as that of the disk depends on the shape of the disk and approximately on its length-breadth ratio, so that cells of different shapes undergo hæmolysis at different critical volumes, which are attained in different tonicities. This permits quantitative explanation of the differences in resistance of red cells to hæmolysis in different species of mammals and in the different red cells of the same animal.

Hypervitaminosis D.

AGNES FAY MORGAN, LOUISE KIMMEL AND NORA C. HAWKINS (*Journal of Biological Chemistry*, August, 1937) have studied the toxicity of different forms of vitamin *D* concentrates in the rat. Toxic effects (depression of growth, mortality, hypercalcæmia, decrease in femur ash and increase in calcium content of the viscera) were most marked in animals given up to 10,000 units of vitamin *D* daily as irradiated ergosterol. The same doses accompanied by large doses of vitamin *A* (up to 10,000 units daily) produced less severe but still obvious symptoms of hypervitaminosis *D*. When similar doses of vitamin *D* were fed in the form of fish liver oil concentrates the toxicity was found to be very much reduced.

Vitamin B_1 in Infancy.

M. R. PRICE (*Archives of Disease in Childhood*, August, 1937) presents a brief review of the literature relating to the amount of vitamin B_1 required by the nursing child. He concludes that the requirement is about 200 units per day and that many nursing mothers do not have sufficient vitamin B_1 in the diet to ensure an adequate supply to their babies. It is considered possible that some infantile complaints and general unthriftiness may be due to deficiency in vitamin B_1.

Food Requirements of Children.

JOHN W. MABONEY AND JOSEPH A. JOHNSTON (*American Journal of Diseases of Children*, July, 1937) observed the basal metabolism, food intakes and nitrogen balances of boys and girls from four to fourteen years old. Gains in weight and height were obtained when the diet supplied about 70% more calories than basal requirement. When calorific intake was adequate for growth, protein intake was found to be optimal when protein supplied 15% of the total calories. Amounts greater than 20% produced harmful effects, while amounts less than 15% frequently resulted in a negative nitrogen balance.

Special Abstract.

THE IMMUNOLOGICAL REACTIONS OF THE FILTERABLE VIRUSES.

A MONOGRAPH on the immunological reactions of the filterable viruses by F. M. Burnet, E. V. Keogh and D. Lush, of the Walter and Eliza Hall Institute, Melbourne, has recently been published as a special number of the *Australian Journal of Experimental Biology and Medical Science*. The object of this monograph was primarily to present in connected form an extensive series of experiments on the inactivation of viruses by immune sera in which the technique of egg membrane inoculation was used as the experimental method. In order to present an adequate discussion of the results so obtained, it seemed necessary to widen the original scope and to include a general review of the problem of the interaction of viruses and antisera. Both plant viruses and bacteriophages, as well as the viruses pathogenic for man and animals, are considered.

The original work included comprises a detailed reexamination of the nature of the bacteriophage-antiphage reaction by Burnet and Lush and quantitative studies of the inactivation on the egg membrane of the following viruses: vaccinia and Newcastle disease of fowls (Keogh), influenza (Burnet), infectious laryngo-tracheitis of fowls and louping ill (Burnet and Lush) and rabbit myxomatosis (Lush). In the discussion of the results of other workers in similar fields, attention is mainly concentrated on the reactions of vaccinia virus, which is that most extensively used in theoretical researches of this type.

The problem of the nature of the interaction between viruses and immune sera has attracted a good deal of interest in recent years. It cannot be said that it is at present a problem of much importance in practical medicine, but there are signs that immunological methods may be used extensively in future attempts to control such virus diseases as yellow fever, measles and influenza. If immunization is to be attempted by means of virus-immune serum mixtures, as seems likely, it is of obvious importance to know as accurately as possible what is the nature of the virus-antibody interaction.

If we are to answer this question, it is first necessary to understand the nature of the two reagents concerned. What are the physico-chemical characteristics of viruses on the one hand and of antibodies on the other? In their preliminary summary of the nature of viruses and bacteriophages, the authors take a frankly biological attitude regarding these agents as minute living micro-organisms, not as autocatalytic proteins or self-perpetuating growth stimulants. With further knowledge it may be possible to consider some of the smaller viruses in physico-chemical terms, but in the present state of science the application of vaguely formulated concepts like autocatalytic protein molecules leads only to confusion. Some of the larger viruses, such as those of psittacosis and vaccinia, are quite certainly small bacterium-like organisms which lack the power to grow and multiply anywhere but in the living cells of a susceptible host. As we descend the scale of size we reach eventually such viruses as those of poliomyelitis and foot and mouth disease. These are very little larger than certain pure protein molecules and there are difficulties in regarding them as fully living organisms. But nowhere in the series, from the smallest virus to the largest, can a line be drawn to separate one group from the other.

Viruses then are regarded as minute microorganisms of size varying from about 0·01μ to 0·3μ, composed, like all living organisms, essentially of protein, and, like bacteria, carrying on their surface active atomic groupings. Some of these surface groups are responsible for antigenic specificity, others for making the contact with structures of the host cell which are a necessary preliminary to intracellular multiplication. Amongst the animal-pathogenic viruses, no technique for the study of this second set of surface groupings is yet available. Bacteriophages, however, become attached to susceptible bacteria by such means, and it is possible to analyse the relationships between the two types of surface "receptors". It is probable that similar relationships hold for other viruses.

The theory of antibodies is now in a fairly complete state. There is no serious objection to the view that antibody molecules are serum globulin molecules which differ from normal globulin in having certain active patches on their surfaces modified so as to "fit" antigenically active groupings on the corresponding antigen. It will be realized that a globulin molecule is large enough to be considered as a spherical structure with a surface large enough to carry several active patches of this sort. The mechanism by which such modified globulin molecules are produced in response to the antigenic stimulus is still unknown. The nature of the interaction between antibody and antigen, however, is relatively well understood. There is a union between the active patch on the globulin molecule and the determinant group on the antigen molecule. Both antigen and antibody molecules will normally have several reactive points on their surfaces, and when reagents are relatively concentrated, cross linkages will result in the formation of a "lattice" structure where each antibody molecule is united to several antigen molecules and *vice versa*.

It is the central theme of the discussion in the present monograph that the immunological reactions of bacteriophages and viruses are qualitatively explicable along the same general lines as have been proved to hold for the interactions of soluble antigens or bacteria with their respective antisera.

The objections which have been raised to this view are derived from certain experimental results, of which the most important are:

1. In many instances neutralization of the virus appears to occur instantaneously.

2. No aggregation or complement fixation reactions can be demonstrated with some viruses. In other instances, although such reactions occur, they seem to be due to other antibodies than those responsible for inactivating the virus.

3. "Neutral" mixtures of virus and serum can readily be reactivated by simple procedures such as dilution in saline solution.

4. A mixture which is non-infective when given to a susceptible animal by one route may be highly infective if given by another route.

In the authors' opinion these difficulties arise mainly from the fact that union between the virus particle and the antibody molecule is a readily reversible one, and is stabilized only when the virus is in sufficient concentration to allow the formation of an aggregate or when a very long time of contact is allowed. Their summarized interpretation of the virus neutralization reaction is as follows.

> 1. Virus inactivation by immune serum results primarily from union of antibody to the virus surface. This union is a reversible one; it takes place at a rate and reaches an equilibrium determined by the ordinary laws of reversible chemical unions.
>
> 2. Union of antibody has no instrinsic inactivating effect on the virus; the inactivation is a result of the interaction between susceptible cell and antibody-coated virus particle. Certain susceptible cells are protected against infection by lesser degrees of antibody coating on the virus particle than are required by other types of cell.
>
> 3. In practice, the experimental results obtained with any given virus will deviate from the theoretical values owing to the operation of one or more of the following factors: (*a*) The wide range of variation in

the virus population in regard to susceptibility to inactivation by serum. (*b*) The time required to reach equilibrium may be greater than that experimentally practicable. (*c*) The lag between the time of inoculation and the initiation of an eventually demonstrable lesion by the primary infection of a susceptible cell may vary, both in regard to the tissue inoculated and from individual to individual. (*d*) Secondary reactions, particularly with concentrated reagents, may occur *in vitro*; aggregation, secondary irreversible or less readily reversible antibody-virus union, and non-specific inactivation by environmental factors. (*e*) When mixtures of virus with relatively large amounts of serum are inoculated, a corresponding degree of local and general passive immunity is produced which may prevent initiated infections developing to demonstrable lesions.

Although there is a general resemblance of the immunological reactions of plant viruses and bacteriophages to those of animal viruses, an important difference is shown by the bacteriophages, and probably also by the plant viruses. Bacteriophage-antibody union is either irreversible or, more likely, reaches an equilibrium point at which a very much smaller proportion of the reagents remain uncombined than is the case with animal virus reactions. This difference led to certain speculations on the biological significance of the immunity mechanism against viruses. We may take the point of view that since both virus and susceptible host have managed to survive in the balanced struggle for existence between them, mutual adaptations directed toward the survival of both are certain to have developed. The authors suggest that the immunological processes can be regarded in this light, and that the ease with which virus-antibody union can be dissociated by dilution is particularly adapted to allow survival of both host and virus. In the partially immunized host toward the end of a virus infection, the circulating antibody will ensure that all virus particles not actually in the cells are united with antibody and in a relatively innocuous condition. Virus liberated into the environment will also in general be associated with, and at least in part united to, antibody. On entering the tissues or body fluids of a new host, however, the antibody-virus union will be dissociated and the virus particles become infective. Such a characteristic would be of distinct survival value to the virus species, and it is suggested that a special type of surface structure adapted to unite with antibody in this easily dissociable fashion has been specially evolved by those viruses infecting animal hosts. Amongst bacteriophages and plant viruses such a mechanism would be of no survival value, and none has developed.

The site of formation of virus antibodies in the body probably depends largely on the type of infection. The question is of particular interest in regard to those viruses which are limited to one particular type of tissue. In human pathology, poliomyelitis virus, which can infect only the cells of the nervous system, and influenza virus, with a similar restriction to the lining of the respiratory tract, are the important examples. In both these diseases there is clear experimental evidence that substantial immunity can be acquired only as a result of infection by the natural route. In both instances antibodies can be produced by suitable subcutaneous injections of virus, but these antibodies are not effective in protecting the animal against infection of the susceptible tissue. Since the immunity acquired by infection is specific, it must equally be due to antibodies, and the difference is probably that the antibody is produced by cells in close anatomical relationship to the infected cells. Such cells retain the potentiality to respond rapidly to a new antigenic stimulus of the same type by the production of antibody, and in this way are probably responsible for the "local" immunity characteristic of these two virus infections.

The monograph concludes with a brief discussion of the principles of active immunization against virus diseases. Since many virus infections are followed by very substantial and prolonged immunity, it is natural to attempt to induce similar immunity by harmless methods. It must be recognized, however, that in all probability no method of artificial immunization will produce a degree of immunity more substantial than that following natural infection. Since an infection with the virus of an ordinary cold leaves no demonstrable immunity, it would be futile to attempt artificial means of inducing such immunity. On the other hand, immunity after an attack of yellow fever probably lasts for life, and present methods of artificial immunization against this disease appear to be remarkably efficient.

Practically all effective methods of immunization make use of living virus and depend on the establishment of a subclinical infection as the immediate stimulus to the production of immunity. To reduce the intensity of the infection to the subclinical level, one of three methods is employed.

1. Living virulent virus is introduced into the body by some route less dangerous than the natural one. Psittacosis virus, for instance, when inoculated subcutaneously, produces only a trivial infection and appears to induce a satisfactory immunity against infection of the lungs.

2. Virus is administered along with a sufficient dose of immune serum to hold the infection in check, but not to abolish it. This has been used mainly in veterinary work where an occasional accidental death is of no great importance. It is usually too dangerous for human applications, but in principle the prophylactic use of measles convalescent serum during the incubation period is equivalent to the method of virus-immune serum immunization. The serum is given about half-way through the incubation period and usually converts the infection into a mild or abortive one which is still capable of providing normal subsequent immunity.

3. The virus used for immunization has been attenuated by long-continued passage on some other animal, or by some method of tissue culture. This is, of course, the classical Jennerian method. It has recently been applied to immunization against yellow fever. In one method, tissue culture virus alone is used. This is now the most popular method, but earlier successful results were obtained by combining the inoculation of modified mouse virus with a dose of human immune serum. Immunization of ferrets and mice against influenza with virus adapted to growth on the egg membrane has been obtained by Burnet, and the method may eventually be applicable to human beings.

In many respects immunization by killed virus would be the ideal, since it would eliminate any possibility of inducing infection in the subject or of rendering him a source of contagion to others. Unfortunately there are very serious difficulties which have so far prevented any success whatever by such methods. When living virus is administered, the actual antigenic stimulus is produced by the multiplication of virus in the body. With dead virus, only the amount actually introduced is available to provoke immunity. In ordinary extracts of infected tissue the virus particles form only a very small proportion of the material present, and it is impracticable to inoculate very large amounts. Modern methods of high speed centrifugation have made it technically possible to obtain a few milligrammes of almost pure virus particles from large volumes of tissue extract, but the cost of such methods would prevent any practical application.

A more important objection still is that any method of killing virus necessarily damages the atomic groupings on the surface of the virus particles and so modifies its antigenicity. It has been shown in fact that killed vaccinia virus does not produce the same degree or quality of immunity in the rabbit that infection by living virus does.

For both practical and theoretical reasons, therefore, it seems unlikely that immunization with killed virus preparations will ever be more than an unimportant subsidiary method for the prophylaxis of virus infections.

British Medical Association News.

MEDICO-POLITICAL.

ANNUAL MEETING OF THE DELEGATES OF THE AFFILIATED LOCAL ASSOCIATIONS OF MEMBERS WITH THE COUNCIL OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

THE annual meeting of the delegates of the Local Associations affiliated with the New South Wales Branch of the British Medical Association was held at the William Henry Crago Council Room, British Medical Association Building, 135, Macquarie Street, Sydney, on October 1, 1937, DR. LINDSAY A. DEY, the President, in the chair.

The following delegates were present: Dr. Kevin Byrne (Canterbury-Bankstown Medical Association), Dr. A. T. Roberts (Central Northern Medical Association), Dr. R. O. Williams (Central Southern Medical Association), Dr. A. M. Gledden (City Medical Association), Dr. B. W. Stevenson (Eastern Suburbs Medical Association), Dr. G. F. L. Elliott (Illawarra Suburbs Medical Association), Dr. B. G. Wade (Kuring-gai Medical Association), Dr. R. J. Jackson (Northern District Medical Association), Dr. J. Coen (North-Eastern Medical Association), Dr. L. Fetherston (South-Eastern Medical Association), Dr. E. L. Newman (Warringah Medical Association), Dr. M. L. Coutts (Western Suburbs Medical Association).

The following members of the Council were present: Dr. George Bell, Dr. K. S. M. Brown, Dr. B. T. Edye, Dr. C. H. E. Lawes, Dr. E. H. M. Stephen, Dr. E. A. Tivey, Dr. A. S. Walker, Dr. G. M. Barron, Dr. A. M. Davidson, Dr. W. K. Inglis, Dr. W. F. Simmons, Dr. A. C. Thomas, Dr. R. C. Traill, Dr. G. C. Willcocks, Dr. J. Adam Dick (Vice-President).

Dr. Hugh Hunter, Acting Medical Secretary, attended.

The Editor of THE MEDICAL JOURNAL OF AUSTRALIA was represented by Dr. Beatrix Durie.

Welcome of Delegates.

Dr. Lindsay Dey, the President, welcomed the delegates. It was, he said, particularly important at the present time that the Council should be able to keep in touch with members who were in suburban and country practice. He appreciated the time spent and the distances travelled by delegates in order to attend the meeting, and was grateful to them for the trouble they had taken in order to be present.

In the past much good work had been done in keeping the local associations in touch with the Council by Dr. A. S. Brydon, Dr. R. J. Jackson and others, who had attended quarterly meetings of the Council as well as the annual meeting of delegates. He commended this practice to delegates. He apologized for the unavoidable absence of Dr. J. G. Hunter, who was in Tasmania on urgent business connected with the British Medical Association in that State. Two vital matters were to be discussed at the present meeting, namely, the question of national insurance and the suggestion of the formation of a medical association in Australia on the lines of the Canadian Medical Association. Voting at this meeting was by delegates only, not by members of the Council.

Metropolitan Admission Depot.

A motion was moved by Dr. B. W. Stevenson and seconded by Dr. E. L. Newman to the effect that it be suggested to the Council that the Hospitals Commission be asked to establish and supervise a metropolitan hospital admission depot to ensure the prompt admission into hospital of patients requiring urgent medical and surgical treatment, so that the risk of fatal issue in the event of delay in admission to hospital should be avoided.

Dr. B. W. Stevenson said that it was sometimes impossible to get into any hospital in Sydney a patient who was suffering from an urgent surgical condition. He described the predicament of a busy general practitioner, with other urgent cases awaiting his attention, who could not obtain admission to hospital for a patient with acute appendicitis. It was often impossible to spend much time ringing up every hospital in Sydney. The Hospitals Commission should be asked to provide an efficient central authority to deal with cases of this kind.

Dr. E. L. Newman suggested that as a last resort the hospital could be made to take the responsibility for refusing admission of such cases by their being sent to hospital by ambulance.

Dr. L. A. Dey said that all general practitioners met occasions on which urgent cases were refused by hospitals. He deprecated Dr. Newman's suggestion. The ambulance authorities would not carry a patient to hospital on such terms. The matter was one for the Hospitals Commission.

Dr. E. H. M. Stephen said that he too had had patients with urgent conditions refused by every hospital in Sydney. There was no reason why any patient who was seriously ill should be refused a bed in hospital; if necessary, hospitals should make emergency arrangements.

Dr. Kevin Byrne said that his association was in agreement with the motion. The Hospital Admission Depot tried to find beds for all urgent cases, but there were times when officials could do nothing. He had on occasions found it necessary to admit into private hospitals patients who were quite unable to pay for medical or hospital attention. He suggested that the Hospitals Commission should pay hospital (not medical) fees in such circumstances.

Dr. E. L. Newman said that the Hospitals Commission had stated in the Press that beds were always available for such urgent cases.

Dr. E. H. M. Stephen thought that it might be suggested to the Hospitals Commission that when no public hospital bed was available, the Commission would be responsible for the nursing fees of a private hospital.

Dr. C. H. E. Lawes related his experience of a case in which he had had to make himself responsible for the payment of private hospital fees for a patient with acute pneumonia, because he was told that no bed was available for her in any hospital in Sydney. He suggested that the Hospitals Commission should provide for a bed in a private hospital until the patient was well enough to be moved to a public hospital. He referred to a country centre where such an arrangement had been made by a local association, the fees being paid by local authorities.

Dr. George Bell referred to the necessity for ringing up a clerk at the Hospital Admission Depot. It was not right that matters of such vital importance should be in the hands of a clerk. A medical practitioner should control the allotting of beds for urgent cases.

Dr. E. A. Tivey and Dr. Hugh G. Hunter also took part in the discussion.

The motion was carried.

Proposed Form of Contract between Medical Officer and Intermediate and/or Private Patient in Public or Community Hospital.

In the absence of Dr. S. H. Dawes it was proposed by Dr. B. G. Wade and seconded by Dr. R. J. Jackson:

> That a uniform form be printed containing a contract between the medical officer and intermediate and/or private patients in community hospitals and that official arrangements be made whereby hospital secretaries obtain the patient's signature to the contract at the time of admission.

Dr. B. G. Wade said that it was sometimes difficult to arrange for the signature of such forms. At present the medical attendant had to see to the matter himself. It was not always possible to see patients or their relatives before the patient was admitted to hospital. He thought it could be arranged that the form be presented at the time of admission to hospital.

Dr. R. J. Jackson said that the motion was to some extent similar to the one which followed it on the agenda

paper, and which dealt with a standard form of contract between medical attendant and patient. Isolated towns had used various forms of contract. He thought it important that a uniform form authorized by the Council should be used throughout the State. He referred to a doctor who sent an injured man into hospital. He saw the patient on two occasions and sent him an account. The insurance company concerned asked the patient whether he had signed a form in regard to the doctor's fee. As he had not done so, the account was not paid, the company quoting Judge White's ruling that when no form was signed the medical attendant had no legal power of recovery.

Dr. Hugh Hunter said that a form of contract between medical officer and intermediate or private patient in hospital had been printed by the Australasian Medical Publishing Company, Limited, and was now available. No special form for this purpose was authorized by the *Public Hospitals Act*.

Dr. L. A. Dey read Section 36 of the *Public Hospitals Act* (1929-1934). He said that the form under discussion should not be confused with forms four and five authorized by the act, which were used by the hospital authorities.

Dr. A. T. Roberts said that forms such as the one being discussed were in use at the Newcastle Hospital.

Dr. L. A. Dey reminded members that the matter under discussion was whether the hospital secretary should obtain the patient's signature on the form.

Dr. B. G. Wade cited the case of a patient with chronic appendicitis who was admitted to Hornsby Hospital as an intermediate patient. He had not seen the patient until some time after admission. In cases such as this, which were not urgent, it would be more convenient for the hospital secretary to see to the signing of the form.

Dr. Kevin Byrne said that it would be difficult for a hospital secretary to intervene in a contract between doctor and patient.

Dr. Hugh Hunter said that a contract was not legally binding unless it was explained to the signatory by a third party.

Dr. L. A. Dey said that unless a fee was mentioned such a contract was not legally binding.

Dr. B. W. Stevenson pointed out that as the course of an illness might be prolonged by unknown factors, it was not possible to assess the medical attendant's fee at the outset.

Dr. R. J. Jackson said that his association would not agree to the proposal that a hospital secretary should sign an agreement concerning the doctor's fee. Such an arrangement would shake the confidence that existed between doctor and patient. His seconding of the motion would have to be *pro forma*, as he did not intend to vote for it.

Dr. R. O. Williams said that in Goulburn the secretary had signed such forms for the past two years. There was no resident medical officer at the hospital, and the arrangement had been eminently satisfactory. The secretary's office was at the hospital.

Dr. L. A. Dey read counsel's opinion on the matter of contract.

Dr. C. H. E. Lawes and Dr. Elliott also spoke.

The motion was not carried.

It was moved by Dr. R. J. Jackson and seconded by Dr. B. G. Wade:

> That it be a recommendation to the Council that it authorize immediately a standard form of contract between medical attendant and patient for the use of members.

Dr. R. J. Jackson read Judge White's ruling on the recovery of fees under the *Public Hospitals Act*. He said that it was only in exceptional cases that it would be necessary to use such a contract in private practice. There were rare occasions when a standard form, authorized by the Council, would be of great help.

Dr. L. A. Dey said that a form of contract was useful in dealing with insurance companies. In his opinion this was its only use.

Dr. George Bell said that the Council should take legal advice as to whether such a contract was binding.

Dr. Keith Brown deprecated the use of a printed form. Such a form was anathema to most patients. When necessary, a typewritten form could be drawn up by the medical attendant.

Dr. Kevin Byrne and Dr. B. W. Stevenson also spoke.

The motion was carried.

Friendly Society Lodge Practice.

Common Form of Agreement.

It was proposed by Dr. Kevin Byrne and seconded by Dr. E. L. Newman:

> That the attention of the friendly societies should be drawn to the form in the common form of agreement whereby it is laid down that payment of cheques to medical officers should be made before the end of the current quarter.

Dr. Kevin Byrne said that members of his association thought that a little slackness had crept into the matter of the payment of cheques by friendly societies.

At the request of Dr. R. J. Jackson, Dr. L. A. Dey read Clauses 24 and 28 of the common form of agreement.

Dr. Hugh Hunter said that grand secretaries of friendly societies would like any delay in payment to be brought to their notice. It was the policy of the societies to pay cheques promptly.

The motion was not carried.

Treatment of Unemployed and Unfinancial Lodge Members.

It was moved by Dr. Kevin Byrne and seconded by Dr. B. G. Wade:

> That, in view of the Government medical service for the unemployed, the hitherto existing arrangement, whereby a medical officer was willing to treat a number of *bona fide* unemployed or unfinancial members equal to 10% of the financial members on that medical officer's list, should be abolished.

Dr. Kevin Byrne said that under the existing arrangement the practitioner was legally bound to attend unemployed persons at any time or provide a substitute. In view of this a recent utterance in Parliament, to the effect that hitherto there had been no arrangement for providing medical attention for poor people, should be contradicted by the Council. Doctors had treated poor persons free for years without the intervention of a third party.

Dr. L. A. Dey said that a statement refuting such assertions had appeared under his name in *The Sun* on Saturday, September 25. The Association was not unaware of such statements, which were constantly cropping up.

Dr. A. T. Roberts said that of late years his list of unfinancial lodge members was dwindling, as unemployment decreased.

Dr. W. F. Simmons and Dr. Keith Brown also spoke.

The motion was carried.

Mass Immunization against Diphtheria.

It was moved by Dr. B. G. Wade and seconded by Dr. R. J. Jackson:

> That the scheme for diphtheria immunization recommended by the Committee of this Association to the Council be put forward for approval by the annual meeting of delegates on October 1, 1937, and that a copy of the scheme be sent to each local medical association.

The scheme was as follows:

1. That this Association is of opinion that intermittent efforts at mass immunization is not a sound method of dealing with the diphtheria problem.

2. That diphtheria immunization should be placed on a sound footing by establishing a permanent system of immunization.

3. That immunization should be conducted as a general practitioner service and that the cost should be paid in part by the State or other public authority and in part by the patient.

4. That the following routine is recommended. (*a*) That the Board of Health, after consultation with the Registrar of Births, should send a notice to the parent or guardian of each child when the child becomes a year old, advising that the child be taken to the doctor for diphtheria immunization. (*b*) That the Education Department should adopt a policy of advising the parents of all children to have their children immunized against diphtheria on sending them to school, unless they have been previously immunized. (*c*) That medical practitioners be given by a public authority anatoxin for immunization free of charge, and that they be allowed 5s. from the State or a public authority and 5s. from the parent or guardian for each child immunized, except in such cases as are certified by the police, relief or other authority to be unable to afford a fee, when the State will pay the whole fee of 10s. (*d*) That the fee from the parent be payable at the time of the immunization, and the fee from the public authority be paid monthly or quarterly, as may be arranged, on the medical practitioner's presentation of his list of children immunized during the period, the list to contain the patient's name and address, date of skin test, and dates and amounts of immunization doses, and signature of parent or guardian.

5. That this Association is of opinion that in this way a satisfactory method of immunization can be established. The cost to public authorities of engaging nurses, purchasing equipment and making arrangements for the use of the halls *et cetera* will be eliminated. The dislocation of the work of municipal staffs will be avoided.

Dr. B. G. Wade said that the present arrangements were unsatisfactory. The councils had endeavoured to foist the work of immunization onto local hospitals. This had led to disagreement between shire councils, hospitals and medical practitioners; and in consequence of this immunization was not likely to be completed. Over 1,500 children in the Kuring-gai Shire had not been immunized and were not likely to be immunized. In his opinion, spasmodic and incomplete methods of immunization were extremely unsatisfactory. The scheme now before the meeting provided for constant and systematic immunization of all children in the community.

Dr. Kevin Byrne said that three doctors, of whom he was one, had immunized the children of the Lakemba Public School. Each child had paid four shillings and it was found that this easily covered the cost of medical fees and anatoxin. Each doctor was paid at the rate of one guinea per hour, and put in approximately ten hours at this work. Over 700 children were immunized. Help in organization, sterilization and clerical work was given by a nurse from the Canterbury District Hospital and by the local health authorities. This arrangement had worked very well.

Dr. C. H. E. Lawes said that the new scheme was a well-thought-out effort to cope with the situation. Mass immunization was too haphazard. He thought that the charges mentioned were somewhat high. Perhaps a fee of four shillings would be sufficient. If the patient was absolutely unable to pay, the doctor should be content with four shillings from the Government.

Dr. R. Traill did not consider the charges excessive. The fee mentioned was too low for private cases, for which a guinea should be charged. This included the cost of anatoxin.

Dr. W. F. Simmons said that the friendly societies had already been notified that the fee for immunization, including cost of anatoxin, was one guinea per child.

Dr. B. G. Wade said that the scheme was not intended to interfere with the fee charged in private cases, but to replace mass immunization.

Dr. H. G. Hunter read a telegram from the Western Districts Association supporting the motion.

Dr. R. J. Jackson also spoke.

The motion was carried.

Honorary Medical Officers to Sporting Bodies.

It was proposed by Dr. G. M. Barron and seconded by Dr. C. H. E. Lawes that:

> No member shall act as honorary medical officer to any racing, sporting or athletic club or to any institution which is carried on for other than charitable purposes.

Dr. A. T. Roberts asked whether an exception might be made in the case of sporting associations which made no charge for admission.

Dr. R. J. Jackson, Dr. George Bell and Dr. Hugh Hunter also spoke.

The motion was adopted.

National Health Insurance.

It was moved by Dr. W. F. Simmons and seconded by Dr. George Bell:

> That the policy of the Association in regard to the following matters relating to health insurance, namely, income limit, medical services to be included, capitation rate and number of insured persons on the list of a medical practitioner, be:
>
> 1. Income limit: three hundred and sixty-five pounds *per annum*.
>
> 2. Medical services to be rendered by a medical practitioner to an insured person should include all proper and necessary medical services other than treatment in respect of a confinement, provided the services rendered do not necessitate the cooperation and/or assistance of any dental or medical practitioner, and other than treatment involving the application of special skill and experience of a kind which practitioners as a class could not reasonably be expected to possess.
>
> 3. Capitation rate: (*a*) one pound *per annum* if medical benefit is available to the insured person only; (*b*) two pounds *per annum* if medical benefit is available to the insured person and dependants.
>
> 4. Maximum number of insured persons allowable on the list of a medical practitioner to be fifteen hundred (1,500).

Dr. L. A. Dey said that the Federal Council had considered national health insurance at its recent meeting in Adelaide and had discussed Sir Walter Kinnear's report. The details of the present motion were based on that discussion. He thought that each clause should be considered separately.

Dr. R. J. Jackson said it was difficult to discuss the clauses separately, as they were interdependent. He was in some doubt about the meaning of the second clause. Was this to be a comprehensive scheme? Was it to include operative work?

Dr. L. A. Dey said that the wording of Clause 2 answered Dr. Jackson's question. The phrase "provided that the services rendered do not necessitate the cooperation and/or the assistance of any medical practitioner" excluded operative work, as the assistance of at least one other practitioner was usually required for this.

Dr. R. J. Jackson quoted some figures sent to him by a practitioner in a country town, who had tabulated the fees received by him. The average amount paid annually by patients *per capita* for usual medical services was £1 12s.; the amount paid *per capita* for all services but confinements was £2 12s.; the amount paid for complete medical service including confinements was £2 18s. If the scheme under discussion came into force it would materially affect this type of practice.

Dr. L. A. Dey said that it should be remembered that the income limit of £365 included 90% of the population, at any rate in the eastern States. He had been told

that miners in Western Australia sometimes earned £20 a fortnight. He personally thought that the income limit should be £312.

Dr. Hugh Hunter read some figures compiled from the *questionnaire* sent to members of the Association. Close on 400 replies had been received; of these, 150 had been satisfactory. The replies received showed a great diversity of opinion.

Dr. Kevin Byrne, on behalf of Dr. Milton Coutts, moved an amendment that the income limit be altered from £365 to £312. This amendment expressed the views of the Western Suburbs Association.

Dr. G. F. Elliott seconded the amendment.

Dr. B. G. Wade asked for Commonwealth statistics relating to the number of persons whose incomes were less than £365.

Dr. Hugh Hunter replied that the income limit of £365 included approximately 90% of the population.

Dr. A. M. Davidson was in favour of an income limit of £365. A lower income limit would mean a lower capitation fee. The whole basis of the scheme was to make it easy for the working man to get medical attention. He thought the medical profession should adopt a generous attitude about the income limit.

Dr. B. W. Stevenson said he thought that a sliding scale would be better. His association thought the limit too high. Two hundred and sixty pounds was enough.

Dr. L. A. Dey thought a sliding scale unnecessary. Wherever the limit occurred there would be hardship. This was inevitable.

Dr. Hugh Hunter read portion of the English contract of service.

Dr. R. J. Jackson inquired what would be considered a high capitation fee.

Dr. A. M. Davidson said that the English capitation fee was 9s. *per annum* per worker; but English conditions were not comparable with Australian conditions. Australian workers demanded and received much higher wages than English workers.

Dr. E. L. Newman inquired why the capitation fee in the case of the friendly societies should be 26s. when the income limit was only £260.

Dr. Thomas said that this scheme would be much more universal than the friendly societies' scheme. Many patients were enthusiastic about it. It was part of the political programme of both parties.

The amendment moved by Dr. Kevin Byrne and seconded by Dr. Elliott was lost.

Dr. Keith Brown also spoke.

The meeting resolved that the income limit should be £365 *per annum*.

The meeting then discussed the second clause of the motion.

Dr. R. J. Jackson agreed that surgery should be omitted from the scheme; otherwise men who did not wish to do surgery would be forced to do it. In Scotland the scheme was quite workable without operative work.

Dr. B. G. Wade agreed with Dr. Jackson. Only a small proportion of practitioners were honorary surgeons in hospitals. Those who were not honorary medical officers of hospitals could not be expected to operate on insurance patients. Dr. Wade thought the scheme should follow the lines of lodge agreements.

Dr. A. M. Davidson said that the mental outlook of patients in Australia and England was totally different. Patients frequently were not averse to paying for operations and other special services. The agreement, he thought, should be along the lines of the friendly societies' agreement.

Dr. Kevin Byrne said that one important drawback of a scheme which included all types of medical attention was that it would be difficult to hand work on to specialists. It was in the interests of the patient to have machinery for sending him to a specialist if necessary. Many fractures required the service of a specialist.

Dr. Hugh Hunter said that under the English scheme the general practitioner could claim a fee for doing special work, provided he could show that he was qualified to do it.

On the motion of Dr. B. G. Wade, seconded by Dr. Keith Brown, the second clause of the motion was adopted.

The capitation rate was then discussed.

Dr. Lindsay Dey said that the South African Medical Association was of the opinion that the capitation rate should be as follows: for those whose income was below £180, 9s. *per annum*; those whose income was between £180 and £365, 13s. 6d. *per annum*. In the British Columbia scheme the capitation rate suggested was approximately £2 *per annum*. This had been turned down by the Government of British Columbia.

It was moved by Dr. R. J. Jackson and seconded by Dr. A. M. Davidson that the following amendment be adopted in place of the third clause of the motion:

> That the capitation rates be as follows: £1 6s. *per annum* (insured person only) and £2 12s. *per annum* (insured person and dependants).

Dr. Jackson said that the reason for the higher capitation rate lay in the fundamental difference between England and Australia. England was a closely settled country; in Australia the population was sparse and scattered. It might be necessary to have different rates for suburban and country practice.

Dr. L. A. Dey said that the present lodge agreement (26s. and 32s.) was not strictly comparable, as lodge patients were selected lives.

Dr. A. M. Davidson referred to the recent failure of an attempt to have the capitation fee raised in England. The court of inquiry went into the question of cars, tires *et cetera*, instead of considering what a medical man should be paid for looking after 1,500 people and what was the income necessary to run a practice. A minimum gross income was a necessity to a medical man if he was to keep his equipment and knowledge up to date.

The South Sydney Medical Association suggested a capitation fee of 52s. 6d.

Dr. Kevin Byrne, on behalf of Dr. Coutts, said that the Western Suburbs Association wished to recommend that the capitation fees be £1 and £2 12s. 6d. respectively.

Dr. Hugh Hunter read a message from the Western District Association recommending a capitation fee of £3, increased by one-third for rural calls. He thought such a figure too high. The meeting must reason out a figure from the present lodge payments. It had been reckoned that for each lodge member a doctor attended 3·2 individuals per year. Much more attention was usually required by wives and families of lodge members than by lodge members themselves.

Dr. W. F. Simmons said that in South Australia the friendly societies had a differential rate. The capitation rates were: females 14s., males 18s., families 36s. It had been computed that 40% of lodge members were single; 60% were married.

Dr. E. L. Newman also spoke.

As a further amendment to clause 3 it was proposed by Dr. Coen, on behalf of the North-Eastern Association, that the capitation rate be £1 10s. for insured persons only and £3 for insured persons and dependants.

There was no seconder; the motion lapsed.

Dr. Milton Coutts, on behalf of the Western Suburbs Association, moved an amendment making the capitation fees £1 and £2 12s. 6d.

There was no seconder; the amendment lapsed.

The amendment moved by Dr. Jackson and seconded by Dr. Davidson was carried.

Dr. W. F. Simmons moved that clause 4 of the motion be adopted. He said that a maximum number of 1,500 insured persons (not families) would mean roughly twenty services per working day.

Dr. R. J. Jackson seconded the adoption of Clause 4. He said that 1,500 was a reasonable figure for the maximum number of insured persons.

Dr. E. L. Newman expressed the opinion that the maximum number should not be fixed until the capitation fee was fixed.

Dr. R. J. Jackson said that the present motion was merely a basis for argument. If the maximum number was to be changed, the capitation fee could be changed too.

Dr. Bell said that the most important matter was the type of service. The Association wished the service to be better than the British service. This could be achieved only by a higher capitation fee and a lower maximum number of patients.

Dr. Kevin Byrne said that a national insurance scheme would increase the cost of running a practice, as there would be a great deal of clerical work to be done. Men who had not needed a secretary might require one. A higher maximum number than 1,500 might be necessary to meet this additional expense.

Dr. Elliott asked whether it had been mooted or decided that medical practitioners would have all this clerical work to do.

Dr. L. A. Dey said that it was inevitable.

Dr. B. G. Wade asked whether the Association was waiting for the Government to approach it with a scheme or was formulating a scheme to submit to the Government.

Dr. L. A. Dey said that the Association was formulating a scheme which could be presented at a moment's notice if necessary. The present motion, if carried, would be submitted to the Council of the New South Wales Branch and sent on to the Federal Council, which would coordinate the schemes received from the different States.

Dr. George Bell said that the Federal Council wanted information, so that if called on to meet the Government, it could have a scheme ready for presentation.

Dr. R. J. Jackson inquired whether it was likely that there would be time for further discussion at the next meeting of delegates.

Dr. George Bell said that the Council did not know. In any case, he thought members should do their best to keep records. The association should have as much information as possible.

The fourth clause of the motion was adopted.

The motion was carried.

It was moved by Dr. R. J. Jackson, seconded by Dr. B. G. Wade:

> That it be a recommendation to the Medical Politics Committee in the formulation of the policy of the Association in regard to the following matters relating to health insurance, namely, mileage rates and rates for night work, that these be the same as those of the common form of agreement.

Dr. R. J. Jackson said that certain practices in the Highlands of Scotland were specially subsidized on account of bad roads. The common form of agreement was a satisfactory basis for discussion of the matter of mileage.

Dr. A. T. Roberts asked whether this matter was not covered by the previous resolution.

Dr. George Bell said that even if it was there was no harm in stressing it. There were some places in the State where a subsidized scheme, such as the Highland scheme, would be applicable.

Dr. A. M. Davidson said that the cost of transport must not be confused with the question of mileage fees. It was not merely a question of the cost of petrol or of the horse-power of a motor car. The mileage fee was necessary to compensate for loss of time, upset of routine, inconvenience, wear and tear *et cetera*.

The motion was carried.

It was moved by Dr. W. F. Simmons and seconded by Dr. George Bell:

> That in view of the possibility of national health insurance being introduced and the necessity for the profession to be in a position to state its case to the Government, members be requested to record their visits and attendances to lodge patients for a period of one year.

Dr. W. F. Simmons said that in England the British Medical Association had been unable to present any figure to the Government. He suggested that members be requested to keep records, commencing from January 1, 1938. He described a simple system of keeping such records.

Dr. A. T. Roberts read a circular issued by the Central Northern Association. Members had been asked to keep records for three months, commencing from October 1. Each member was asked to record: (i) How many contributors he had. (ii) How many persons he was under contract to treat. (If necessary, lodge members were to be circularized to obtain this information.) (iii) How many attendances, services *et cetera* were performed during this period. (iv) His income during this period.

There ensued a discussion as to the machinery by which this motion was to be carried out.

Dr. B. G. Wade thought that each delegate should ask his own association. This would be more impressive than circulars.

Dr. L. A. Dey thought that members should be approached by delegates and sent circulars also.

Dr. R. J. Jackson suggested that the simplest possible form of circular should be sent from headquarters, followed by local circulars backing it up.

The motion was carried.

Proposal to Form an Australian Medical Association.

It was proposed by Dr. W. F. Simmons and seconded by Dr. Keith Brown:

> That the time has arrived to explore the possibility and desirability of forming an Australian medical association affiliated to the British Medical Association.

Dr. R. J. Jackson said that this matter was so far-reaching that it was difficult for local associations to assess the repercussions of any change. A similar proposal had been made six years ago and had been rejected. He well remembered Dr. Crago saying that it would be a sad day for all concerned if the present arrangements were terminated. He would like to hear something of the standard of the Canadian Medical Association and of its experience since its formation. He thought much more information was required before the matter could be discussed.

Dr. L. A. Dey said that at one time there were two associations in Canada, one of which was a branch of the British Medical Association, the other a Canadian association. The former had retired from the field in order that the two associations could be affiliated under the name of the Canadian Medical Association.

Dr. Hugh Hunter read an account of the formation of the Canadian Medical Association.

Dr. A. M. Davidson spoke against the motion. He agreed with Dr. R. J. Jackson that more information was needed before the matter could be discussed. There was no need for a discussion of a new association; members should make an attempt to obtain autonomy within the British Medical Association. The legal expenses of forming a new association would be considerable, even if the profession were unanimous. If there was no unanimity, anything might happen. The very change of name might be fatal from a business point of view, and at a time when unanimity was most important. He thought it a pity that the matter had been discussed at the Federal Council meeting without notice.

Dr. L. A. Dey stated that the fact that the matter had been discussed in Adelaide necessitated its being brought to the notice of the New South Wales Branch. It was felt that the present was an opportune time to do this.

Dr. Keith Brown said that all members realized that the present time was a critical one. It was essential to

have an efficient body with adequate funds. Unanimity was essential. Efficiency was largely a matter of finance. If the profession would agree to the raising of the British Medical Association subscription the whole thing would be simple.

Dr. B. G. Wade said he was perfectly confident that members would be willing to pay an increased subscription if it was necessary.

Dr. George Bell said that he himself was opposed to the formation of a new association. He thought the constitution of the Federal Council might be a matter for discussion. There was a great deal of goodwill in the name "British Medical Association". To weaken this would do great harm both in England and in Australia.

Dr. W. J. Simmons said that the New South Wales Branch had had some difficult times and had come through them very well. A branch that could put £27,000 into the British Medical Association Building could certainly find £1,000 *per annum* if this was necessary.

Dr. B. W. Stevenson thought that there was no sufficient reason to alter the present arrangement.

Dr. Kevin Byrne also spoke.

It was resolved, on the motion of Dr. R. J. Jackson, seconded by Dr. Kevin Byrne:

> That this meeting is strongly of the opinion that no change should be made in the constitution of the New South Wales Branch of the British Medical Association.

Dr. Keith Brown suggested that the matter be taken back to the local associations. The Council could have an expression of the opinion by the end of the year.

Dr. W. J. Simmons said that there might be some fear of levies.

Dr. George Bell said that the increase need be only 10s. *per annum*. An income of £2,000 would be ample for present expenses. Members must learn to think federally. It was of great assistance to have a federal secretary.

It was resolved, on the motion of Dr. R. J. Jackson, seconded by Dr. Fetherstone:

> That the Federal Council be informed that the time has arrived to discuss a levy to be used in the furtherance of the work of the Federal Council.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Dance, George Barrington, M.B., B.S., 1933 (Univ. Sydney), Ocean Island, Central Pacific.

The undermentioned has applied for reelection as a member of the New South Wales Branch of the British Medical Association:

Wilkinson, Jeffery Wilmott, M.B., B.S., 1914 (Univ. Melbourne), Swift Street, Albury.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Clarke, Colin Woolner, M.B., B.S., 1935 (Univ. Melbourne), Bothwell.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Hayes, Patrick Gavan, M.B., B.S., 1937 (Univ. Melbourne), "Rosenea", Campbellfield.

Kaye, Myer, M.B., B.S., 1935 (Univ. Melbourne), 72, Princess Street, Kew, E.4.

Public Health.

A SCALE OF RATIONS.

THE following letter has been received by the South Australian Branch of the British Medical Association from the Minister of Industry and Employment of South Australia, in acknowledgement of the Government's indebtedness to the committee appointed by the Branch to draw up a scale of rations.

South Australia.
Department of Industry.
Education Building,
Flinders St., Adelaide,
8th September, 1937.

The Secretary,
British Medical Association
(South Australian Branch),
178, North Terrace,
Adelaide.

Dear Sir,

On behalf of the Government of South Australia I desire that you will convey to the members of the British Medical Association who have made such complete and painstaking enquiry into the matter of the adult rations, the thanks of the Government of this State. We realize what a great amount of time the Members of the Committee have devoted to this enquiry, and what a very difficult work it has been. We greatly appreciate the voluntary service which the Members have rendered to the Government and the community.

Yours faithfully,
(Signed) S. W. JEFFRIES,
Minister of Industry and Employment.

Obituary.

JOHN JOSEPH EGAN.

WE regret to announce the death of Dr. John Joseph Egan, which occurred on October 26, 1937, at Sydney, New South Wales.

Corrigenda.

WE are informed that an error has occurred in the issue of the journal of October 16, 1937, at page 686, in the first line of the second column. Speaking of anæsthetic fatalities in the Adelaide Children's Hospital, Dr. E. Couper Black is reported as having dealt with the period "October 1, 1931, to September 30, 1932". This should read: "October 1, 1931, to September 30, 1936".

An error has occurred in the issue of the journal of October 23, 1937, at page 746, line 31. Speaking of tuberculosis of the larynx at the Adelaide congress, Dr. R. M. Glynn is reported to have stated that he had seen three cases in which he had advised tonsillectomy while the patients had evident tuberculosis of the larynx. Dr. Glynn actually stated that he had seen three patients who had been advised to have their tonsils removed when in fact they were suffering from tuberculosis of the larynx.

Diary for the Month.

Nov. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 9.—Tasmanian Branch, B.M.A.: Branch.
Nov. 10.—Victorian Branch, B.M.A.: Branch.
Nov. 12.—Queensland Branch, B.M.A.: Council.
Nov. 16.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 16.—Tasmanian Branch, B.M.A.: Council.
Nov. 17.—Western Australian Branch, B.M.A.: Branch.
Nov. 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 24.—Victorian Branch, B.M.A.: Council.
Nov. 25.—New South Wales Branch, B.M.A.: Branch.
Nov. 25.—South Australian Branch, B.M.A.: Branch.
Nov. 26.—Queensland Branch, B.M.A.: Council.
Dec. 1.—Western Australian Branch, B.M.A.: Council.
Dec. 2.—South Australian Branch, B.M.A.: Council.
Dec. 7.—Tasmanian Branch, B.M.A.: Council.
Dec. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 7.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments.

Dr. F. F. Pincus has been appointed Acting Manager of the Mount Isa Hospital, Mount Isa, Queensland.

* * *

Dr. Harvard Northcroft Merrington has been appointed Acting Government Medical Officer at Mount Isa, Queensland.

* * *

Dr. W. E. Audley has been appointed Deputy Medical Superintendent, Department of Mental Hospitals of New South Wales.

* * *

Dr. E. F. Harbison has been appointed, pursuant to the provisions of the *Workers' Compensation Acts* of Victoria, a Certifying Medical Practitioner at Melbourne, Victoria.

* * *

Dr. J. E. McMahon has been appointed Medical Officer of Health to the Kellerberrin District Road Board, under the provisions of *The Health Act*, 1911 *to* 1935, of Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xviii to xxi.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer.
BALLARAT AND DISTRICT BASE HOSPITAL, BALLARAT, VICTORIA: Resident Medical Officers.
CAIRNS HOSPITALS BOARD, CAIRNS, QUEENSLAND: Medical Superintendent.
CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
DIRECTOR-GENERAL OF HEALTH, SYDNEY, NEW SOUTH WALES: Visiting Medical Officer.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Resident Medical Officer.
HOBART GENERAL HOSPITAL, HOBART, TASMANIA: Resident Medical Officers.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Resident Medical Officers.
ROYAL MELBOURNE HOSPITAL, VICTORIA: Honorary Physiotherapist.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Relieving Assistant Orthopædic Surgeon.
TAMWORTH BASE HOSPITAL, TAMWORTH, NEW SOUTH WALES: Resident Medical Officer.
THE UNIVERSITY OF MELBOURNE, VICTORIA: Part-Time Lectureships in Surgery, Senior Lectureship in Anatomy
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistant.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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